HIGH THROUGHPUT FUNCTIONAL GENOMIC SCREENING METHODS FOR OSTEOARTHRITIS

FIELD OF THE INVENTION

The present invention provides novel functional genomic screening methods for identifying genes and gene products that are involved in OA. Genes and gene products are also provided that have been identified in such screening assays and which are useful *inter alia* as drug targets for treating OA. Methods of treating and diagnosing OA and compositions therefor which use genes and/or gene products identified in these screening assays are also provided.

BACKGROUND

Osteoarthritis (OA) is primarily a non-inflammatory disease characterized by pain and stiffness of the joints caused by the progressive loss of articular cartilage. OA is among the most common age associated disease and is estimated to affect about 56 million individuals worldwide or 80% of the population greater than 60 years old. Although OA is generally considered a degenerative disorder, the disease is associated with activation of chondrocyte cells, the major cell type present in normal articular cartilage. Hallmarks of this cell activation include hypertrophy, proliferation, dedifferentiation, degradation of the existing extracellular matrix, and finally apoptosis.

The molecular etiology of OA remains unknown. Current therapeutic methods for treating OA are therefore directed toward symptomatic relief such as reducing joint pain and secondary inflammatory changes rather than toward treating the disease's underlying causes. Pharmacological interventions that prevent disease progression are not currently available. Many patients thus progress to advanced stages of the disease where total joint replacement surgery is necessary. For reviews, see Pritzken, "Pathology of Osteoarthritis" in *Osteoarthritis* (Brandt *et al.*, Eds.) Oxford University Press 1998, pages 50-61. See also, Sandell & Aigner, *Arthritis and Rheumatism* 2001, 3:107-113.

Large scale sequencing of OA cDNA libraries has identified several putative gene products that are expressed by diseased chondrocyte cells. See, Stokes *et al.*, *Arth. Rheum.* 2002, 46:404-419; Hu *et al.*, *J. Biol. Chem.* 1998, 51:34406-34412; Aigner *et al.*, *Arth. Rheum.* 2001, 44:2777-2789. However, functional information is not presently available for these gene products and their role in OA, if any, remains unknown. The molecular basis of OA therefore remains unknown and only a very limited number of potential drug targets is known. There remains a need, therefore, for therapeutic compounds and methods to treat OA and related diseases. There is moreover a need for novel genes and gene products that may be useful, *e.g.*, as drug targets for such therapeutic methods to treat OA.

In order to identify genes associated with OA that can serve as suitable drug targets, Applicants disclose herein several high throughput screening methods that may be used successfully with chondrocytes. Identification of genes that are critical in mediating the diseased phenotype requires development of comprehensive highly sensitive cell-based assays compatible with high-throughput settings. The availability of methods to shuttle full length cDNA clones from one vector into another (Gateway system, Invitrogen, Carlsbad, CA) combined with the ability to express genes in high levels in disease relevant primary cells using viral vectors and the availability of methods for assay miniaturization and liquid handling have lead to the possibility of efficiently screening for inducers of OA phenotype on a genome wide scale.

Using said methods, Applicants have identified several genes (referred to herein as "candidate genes") in chondrocytes that are associated with OA. Thus, according to the present invention, it is now proposed that these genes and gene products have a role in OA pathogenesis and it is contemplated herein that any one or more of them are useful drug targets for the development of therapeutics for the prevention, treatment or amelioration of OA or related conditions associated with abnormal cartilage degradation.

The invention also provides a method for identifying modulators (e.g. inhibitors) of these newly identified OA related genes and the use of such modulators for the treatment,

prevention, or amelioration of this disease and related conditions, in human and veterinary patients. The invention also provides pharmaceutical compositions comprising said modulators.

SUMMARY OF THE INVENTION

The present invention provides high throughput functional genomic screening (HTS) assays that may be used to identify genes and gene products associated with OA. In preferred embodiments, a HTS assay of the invention comprises steps of transfecting a cell (preferably a chondrocyte cell) with a nucleic acid to be tested in the screening assay (*i.e.*, a "test" nucleic acid) so that the test nucleic acid is expressed by the cell. The transfected cell is then assayed for one or more characteristics that are associated with OA. For example, in one preferred embodiment, a screening assay of the invention comprises steps of detecting expression by the cell of one or more genes or gene products whose expression is known to be associated with OA.

Similarly, screening assays of the invention can be used to identify polypeptides and other gene products that are associated with OA in cells. Such methods involve transfecting a cell (preferably a chondrocyte cell) with a nucleic acid that encodes a polypeptide or other gene product to be tested in the screening assay (*i.e.*, a "test" polypeptide) so that the test polypeptide is expressed by the cell. The transfected cell is then assayed for one or more characteristics that are associated with OA. For example, in one preferred embodiment a screening assay of the invention comprises steps of detecting expression by the cell of one or more genes or gene products whose expression is known to be associated with OA.

A variety of known genes and gene products associated with OA are provided in the application and can be used in the above-described assays. Preferred genes and gene products that are associated with OA (or an "OA phenotype" include, for example, an Aggrecanase-1 gene, an MMP-13 gene, genes of Collagen Types I, IIa and X, an iNOS gene, an Aggrecan gene or gene product, and a Decorin gene, as well as gene products encoded by

any of these genes. Still other genes or gene products that are associated with an OA phenotype and can be used in the methods described here include new marker genes C17, SMOC2, OSF-2, MARCKS, retinoic acid receptor beta, Zic1, BASP1 and DIM1 genes and their gene products which were identified by computational analysis of OA cDNA libraries.

In another aspect, the Applicants have discovered that genes and gene products for an OA phenotype may be rapidly screened by identifying gene and gene products that induce the proliferation of chondrocyte cells. Hence, the invention also provides, in another aspect, a method for identifying a nucleic acid that induces an OA phenotype by transfecting a chondrocyte cell with a candidate nucleic acid, and detecting proliferation of the chondrocyte cell (*e.g.*, by identifying clusters of clonally proliferating chondrocyte cells in cell culture). Similarly, the invention provides methods for identifying a polypeptide that induces an OA phenotype in cells, by transfecting a chondrocyte cell with a nucleic acid that encodes a candidate polypeptide, and detecting proliferation of the chondrocyte cell (*e.g.*, by identifying clusters of clonally proliferating chondrocyte cells in cell culture). In such methods, proliferation of the chondrocyte cells indicates that the candidate nucleic acid or polypeptide is a nucleic acid or polypeptide that induces an OA phenotype.

Genes and gene products that are identified by such screening methods are useful, *inter alia*, for the diagnosis and treatment , prevention and/or amelioration of OA. For example, candidate genes and gene products identified by these screening methods may be used in still other screening assays, to identify compounds that bind to and/or inhibit expression of these candidate genes and gene products. The compounds (i.e.,modulators) identified in these screening assays are useful, *e.g.*, in therapeutic methods for treating OA and as pharmaceutical compositions or medicaments that can be administered in such therapeutic methods. Thus the present invention also pertains to the use of these genes, gene products, compounds and modulators in the manufacture of a medicament and/or as a pharmaceutical for the treatment, prevention and/or amelioration of OA and other cartilage-related diseases.

In still other embodiments, the invention provides methods for treating, preventing and/or ameliorating OA in an individual, by administering an effective amount of a compound that can modulate (i.e. a "modulator") a candidate gene identified by the assay and methods of the present invention. In a preferred embodiment, the modulator inhibits a candidate gene disclosed in Tables V or VI disclosed herein. The invention also provides pharmaceutical compositions that comprise an effective amount of a modulator to a candidate gene identified herein.

Thus, in another aspect, the invention relates to a method to treat , prevent or ameliorate OA, comprising administering to a subject in need thereof a pharmaceutical composition comprising an effective amount of a modulator of a candidate gene and/or ligand thereo (i.e a gene provided in Tables V or VI provided herein. In various preferred embodiments, said pharmaceutical composition comprises one or more modulators to any one or more of said candidate genes and/or ligands thereof.

In another aspect, the invention relates to a pharmaceutical composition comprising a modulator of a candidate gene and/or ligand thereof in an amount effective to treat, prevent or ameliorate OA in a subject in need thereof wherein said modulator, e.g., can inhibit the activity, expression of or ligand binding to, any one or more of the candidate genes disclosed herein e.g., a candidate gene provided in Tables V or VI herein. In one embodiment, said pharmaceutical composition comprises any one or more substances selected from the group consisting of antisense oligonucleotides, triple helix DNA, siRNA, ribozymes, RNA aptamers or double or single stranded RNA directed to a nucleic acid sequence of a candidate gene or ligand thereof wherein said substances are designed to inhibit gene expression of said family member or ligand. In a further embodiment, said pharmaceutical composition comprises antibodies to a candidate gene or ligand thereof, or fragments thereof, wherein said antibodies can, e.g., inhibit the activity of said member and/or ligand.

In yet another aspect of the present invention there are provided assay methods and kits comprising the components necessary to detect expression of polynucleotides encoding a candidate gene or ligand thereof, or polypeptide levels of said candidate genes or ligands

thereof, or fragments thereof, in biological samples derived from a patient, such kits comprising, e.g., antibodies that bind to said polypeptides, or to fragments thereof, or oligonucleotide probes that hybridize with said polynucleotides. In a preferred embodiment, such kits also comprise instructions detailing the procedures by which the kit components are to be used.

The present invention also provides methods for identifying individuals who have OA. Such diagnostic methods involve detecting a candidate gene or gene product (identified by one of the high throughput functional assays described, *supra*) in a biological sample (e.g., chondrocyte cell or cartilage tissue sample) from the individual. Elevated expression of the candidate gene or gene product in the chondrocyte cell or cartilage tissue indicates that the individual does have OA.

The invention also provides methods for identifying compounds that may be used to treat OA. In a first embodiments, these methods involve contacting a test compound to a candidate gene or gene product under conditions sufficient to allow the test compound to bind to a candidate gene or gene product of the invention, and detecting complexes of the test compound bound to that candidate gene or gene product. The detection of the test compound bound to the candidate gene or gene product identifies the test compound as a compound that can be used for treating OA.

In another embodiment, methods for identifying compounds that may be used to treat OA involve contacting a test compound to a cell that normally expresses a candidate gene or gene product of the invention, and detecting expression of that candidate gene or gene product by the cell once it has been contacted with the test compound. In such embodiments, a decreased expression of the candidate gene or gene product by the cell in the presence of the test compound indicates that the test compound is a compound that can be used to treat OA.

DETAILED DESCRIPTION

As used herein and in the appended claims, the singular forms "a", "an", and "the" include plural reference unless the context clearly dictates otherwise. Thus, for example, reference to the "antibody" is a reference to one or more antibodies and equivalents thereof known to those skilled in the art, and so forth.

"Nucleic acid sequence", as used herein, refers to an oligonucleotide, nucleotide or polynucleotide, and fragments or portions thereof, and to DNA or RNA of genomic or synthetic origin that may be single or double stranded, and represent the sense or antisense strand.

As used herein, "high throughput" refers to an increase in screening capacity compared to conventional methods. It is contemplated herein that the high throughput method of the present invention is preferably carried out using microtiter plates (i.e. 96, 384 or 1536 well plates). Assays at a genomic level are also contemplated.

cDNA libraries for use with the high throughput screen disclosed herein are those wherein each cDNA is defined and arrayed in a specific order in high throughput format (multititer dishes). While the examples in the present invention describe results obtained with a proprietary cDNA collection, suitable cDNA libraries are commercially available, for example, from Invitrogen (Carlsbad, CA), Origene (Rockville, MD) as well as the NIH (i.e., the Mammalian Gene Collection).

The term "antisense" as used herein, refers to nucleotide sequences which are complementary to a specific DNA or RNA sequence. The term "antisense strand" is used in reference to a nucleic acid strand that is complementary to the "sense' strand. Antisense molecules may be produced by any method, including synthesis by ligating the gene(s) of interest in a reverse orientation to a viral promoter which permits the synthesis of a

complementary strand. Once introduced into a cell, this transcribed strand combines natural sequences produced by the cell to form duplexes. These duplexes then block either the further transcription or translation. The designation "negative " is sometimes used in reference to the antisense strand, and "positive" is sometimes used in reference to the sense strand.

"cDNA" refers to DNA that is complementary to a portion of messenger RNA (mRNA) sequence and is generally synthesized from an mRNA preparation using reverse transcriptase.

As contemplated herein, antisense oligonucleotides, triple helix DNA, RNA aptamers, ribozymes, siRNA and double stranded RNA are directed to a nucleic acid sequence such that the nucleotide sequence chosen will produce gene-specific inhibition of gene expression. For example, knowledge of a nucleotide sequence may be used to design an antisense molecule which gives strongest hybridization to the mRNA. Similarly, ribozymes can be synthesized to recognize specific nucleotide sequences of a gene and cleave it (Cech. J. Amer. Med Assn. 260:3030 (1988)). Techniques for the design of such molecules for use in targeted inhibition of gene expression is well known to one of skill in the art.

The individual candidate gene products, (i.e. proteins/polypeptides) referred to herein include any and all forms of these proteins including, but not limited to, partial forms, isoforms, variants, precursor forms, the full length protein, fusion proteins containing the sequence or fragments of any of the above, from human or any other species. Protein homologs which would be apparent to one of skill in the art are included in this definition. It is also contemplated that the term refers to proteins isolated from naturally occurring sources of any species such as genomic DNA libraries as well as genetically engineered host cells comprising expression systems, or produced by chemical synthesis using, for instance, automated peptide synthesizers or a combination of such methods. Means for isolating and preparing such polypeptides are well understood in the art.

The terms "sample" or "biological sample" as used herein, are used in their broadest sense. A biological sample from a subject may comprise blood, urine or other biological material with which protein activity or gene expression may be assayed. A biological sample may include, for example, cells, cartilage, blood, tumors or other specimens from which total RNA may be purified for gene expression profiling using, for example, conventional glass chip microarray technologies such as Affymetrix chips, RT-PCR or other conventional methods.

As used herein, the term "antibody" refers to intact molecules as well as fragments thereof, such as Fa, $F(ab')_2$, and Fv, which are capable of binding the epitopic determinant. Antibodies that bind specific polypeptides can be prepared using intact polypeptides or fragments containing small peptides of interest as the immunizing antigen. The polypeptides or peptides used to immunize an animal can be derived from the translation of RNA or synthesized chemically, and can be conjugated to a carrier protein, if desired. Commonly used carriers that are chemically coupled to peptides include bovine serum albumin and thyroglobulin. The coupled peptide is then used to immunize an animal (e.g., a mouse, a rat or a rabbit).

The term "humanized antibody" as used herein, refers to antibody molecules in which amino acids have been replaced in the non-antigen binding regions in order to more closely resemble a human antibody, while still retaining the original binding ability.

A "therapeutically effective amount" is the amount of drug sufficient to treat, prevent or ameliorate pathological conditions associated with OA.

"Subject" or "individual" refer to any human or nonhuman organism.

The high throughput assay disclosed herein is preferably used or performed in an at least substantially automated setting. A multiwell format is suited for performing at least part of the methods of the present invention, but can be performed on many different scales, including screening cDNAs on a genomic scale. The term "automated" as used herein means

able to perform the predetermined steps of the method without, for the most part, requiring manual intervention during the process. In this regard, machines for use in the high throughput methods disclosed herein include, but are not limited to, machines for preparing DNA plasmid preparations, reading DNA concentration and yield, plating cells, automated pipeting stations and luminescence detectors. Such machines are commercially available and familiar to one of skill in the art, for example, the Quiagen 8000 for automated DNA production (Qiagen Inc, Valencia CA), the Beckman Coulter BiomekFX for automated pipetting and transfections (Beckman Coulter, Fullerton CA) and the Fluoroskan Ascent for fluoroscent and luminescent assay readouts (Thermo Labsystems, Franklin, MA).

Nucleic acid transfer into cells (e.g. transfection) may be performed according to any conventional method familiar to one of skill in the art. As mentioned above, transfections are preferably implemented in an automated, multiwell, high throughput format, for example, using commercially available robotics such as a Beckman Coulter BiomekFX.

The present invention provides high throughput screening (HTS) assays that are useful, *inter alia* for identifying therapeutic agents to treat and/or diagnose disorders such as osteoarthritis (OA) that affect the growth and/or degradation of cartilage. In particular, the Examples *infra* describe particular, preferred embodiments of screening assays that identify genes and gene products associated with OA. The genes and gene products identified in such screening assays are therefore useful, *e.g.*, as drug target candidates for the development of novel drug therapies to treat OA and other such cartilage disorders. For convenience therefore, the genes and gene products identified in screening assays of the present invention are generally referred to in this document as "candidate" genes and "candidate" gene products, respectively.

Generally speaking, the HTS assays of this invention allow a user to rapidly screen large numbers of genes, *e.g.*, in a cDNA library, to identify ones that are involved in OA. Briefly, nucleic acids (preferably cDNA molecules) corresponding to the genes to be tested in a screening assay are first transferred to expression vectors that are capable of expressing those "test" genes or gene products in chondrocyte cells. Preferred expression vectors are

retroviral vectors (such as those described in the Examples, *infra*) or other vectors that are capable of expressing the candidate genes at high levels in chondrocyte cells.

Chondrocyte cells are then transformed with the expression vectors carrying these test genes and are assayed for one or more characteristics that are associated with OA. For convenience, such characteristics are generally referred to in this application as "OA phenotypes." However, it is understood that a characteristic assayed or tested for in these screening assays may be any feature that is associated with OA.

For instance, Example 1 describes one preferred embodiment of a HTS assay that uses RT-PCR to measure the expression of one or more genes whose expression in chondrocyte cells is associated with OA. Examples of such genes which are preferred in these methods include Aggrecanase-1 and MMP-13 (the expression of which is associated with cartilage degradation), Collagen Type I, Collagen Type IIa and Collagen Type X (the over expression of which is associated with aberrant chondrocyte cell differentiation such as hypertrophy and proliferation), genes and gene products that induce inflammation (for example, iNOS and Cox-2), and genes such as Aggrecan and Decorin that modulate synthesis or repair of the cartilage matrix.

Such genes, whose expression or, more particular, over expression is indicative of OA in chondrocyte cells, are generally referred to here as "marker genes." However, "marker genes" that may be used in screening assays of the invention are not limited to the particular genes described, *e.g.*, in the examples (see, for example, in Table I or Table II, *infra*). Any gene or gene product whose elevated expression in chondrocyte cells is associated with OA may be used as a marker gene in screening assays according to the present invention. For example, and as explained in further detail below, the screening assays of this invention identify other genes and gene products whose elevated expression is associated with OA. Hence, a candidate gene or gene product identified in such screening assays (for example, any of the candidate genes and gene products listed in Tables V and VI *infra*) may itself be used as a marker gene in another screening assay according to this invention.

Similarly, those who are skilled in the art will appreciate that marker genes which can be used in screening assays of this invention are not limited to gene whose over expression is associated with OA. In particular, a screening assay of the present invention can also use marker genes that are underexpressed (*i.e.*, their expression is reduced) in OA chondrocytes. In such embodiments, the HTS assays of this invention will identify candidate genes that, when expressed in chondrocyte cells, cause the reduced expression of one or more marker genes.

The HTS assays of this invention also are not limited to embodiments that measure the expression of marker genes or their gene products. Other characteristics or phenotypes associated with OA can also be measured or observed, and then used to identify candidate genes in a screening assay. For instance, Example 2 *infra* describes an alternative embodiment of the screening assay which identify cDNAs that induce a particular type of cell proliferation characteristic of OA chondrocytes. In particular, whereas normal chondrocyte cells have a low division rate when grown in a 3-dimensional matrix (*e.g.*, of agarose or alginate), OA chondrocyte cells (both in cell culture and in OA cartilage tissue) grow in clusters of rapidly proliferating chondrocyte cell clones. Accordingly, screening assays of the invention can also identify genes and gene products which, when expressed in chondrocyte cell cultures, cause the formation of such clusters of chondrocyte cell clones.

Genes and gene products that are tested in a screening assay of the invention may be from any source and obtained by any method known in the art. For example, cDNA libraries may be derived from a cell or cell line of interest, which is preferably a chondrocyte cell. Methods for obtaining such cDNA libraries are well known in the art. See, for example, Sambrook *et al.*, *Molecular Cloning: A Laboratory Manual*, Second Edition (1989) Cold Spring Harbor Laboratory Press, Cold Spring Harbor, New York); Glover, D.M. 3ed., 1985, *DNA Cloning: A Practical Approach*, MRL Press, Ltd. Oxford U.K. Vols. I and II). See also, in the Examples, *infra*. Alternatively, however, the genes and Gene Products may be hand selected. For instance, Example 1 describes an embodiment where the genes in a cDNA library are first "datamined" to identify genes and gene products that are particularly useful as drug targets (*e.g.*, for therapeutic compounds to treat OA). Examples of such preferred test

genes are genes that are involved in signal transduction and/or proteolysis (such as receptors, kinases and proteases).

Candidate genes and gene products that are identified in screening assays of the present invention are useful, *inter alia*, as new marker genes for identifying osteoarthritic cells (*i.e.*, cells that are present in cartilage from patients having OA and/or which exhibit one or more characteristics associated with OA). Moreover, the genes and gene products identified in these screening assays can also be used in diagnostic and prognostic applications. Hence, the candidate genes and gene products that are identified in the screening assays provided here can be used to identify individuals who have a disorder, such as OA, that is associated with abnormal cartilage growth and/or repair.

The candidate genes and gene products identified in screening assays of this invention can also be used in prognostic applications to identify individuals who are either have OA or who are at an increased risk of developing OA. Hence, the invention also provides therapeutic methods for treating OA related disorders in individuals. Such methods involve administering a compound to an individual that inhibits the expression or activity of a candidate gene identified in a screening assay of the invention or, alternatively, a compound that inhibits the expression or activity of a candidate gene product identified in a screening assay of this invention.

Various applications and uses for candidate genes and gene products identified in the present invention are described, in detail, *infra*. In particular, the following sections first describe various homologs and analogs of both candidate genes and candidate genes products that can be used in such prognostic, diagnostic, and therapeutic assays. Particular utilities for these candidate genes and gene products (including the various homologs and analogs thereof) are then also described in detail. Finally, the Examples describe detailed, exemplary embodiments of screening assays that are considered part of the present invention. These examples also provide Tables identifying the nucleotide and amino acid sequence (by GenBank Accession number) of both genes and gene products that are identified in such

screening assays. These nucleotide and amino acid sequences are therefore considered examples of preferred embodiments of candidate genes and gene products of the invention.

The present invention may employ a variety of conventional techniques in the arts of molecular biology, microbiology and recombinant DNA technology. Such techniques are well known in the art and are explained fully in the literature. See, for example, Sambrook, Fitsch & Maniatis, *Molecular Cloning: A Laboratory Manual*, Second Edition (1989) Cold Spring Harbor Laboratory Press, Cold Spring Harbor, New York (referred to herein as "Sambrook *et al.*, 1989); *DNA Cloning: A Practical Approach* Volumes I and II (D.N. Glover *et al.* 1985); *Oligonucleotide Synthesis* (M.J. Gait ed. 1984); *Nucleic Acid Hybridization* (B.D. Hames & S.J. Higgins, eds. 1984); *Animal Cell Culture* (R.I. Freshney, ed. 1986); *Immobilized Cells and Enzymes* (IRL Press, 1986); B.E. Perbal, *A Practical Guide to Molecular Cloning* (1984); F.M. Ausubel *et al.* (eds.), *Current Protocols in Molecular Biology*, John Wiley & Sons, Inc.

Candidate polypeptides:

It is understood that, as used in the description of this invention, the term "candidate polypeptide" refers to the polypeptide encoded by a candidate gene of the invention. For convenience, candidate genes and gene products of the present invention are frequently identified here by SEQ ID number and by the GenBank Accession Number(s) for preferred nucleotide or amino acid sequences. However, it is understood that the candidate genes and gene products of this invention are not limited to these particular sequences, but also include homologs and variants evident to one of ordinary skill in the art.

As an example, and not by way of limitation, candidate gene product polypeptides of the present invention include not only polypeptides having the exemplary full length amino acid sequences specified here, but also include polypeptides comprising an amino acid sequence for one or more epitopes or domains of a full length candidate gene product polypeptide. An epitope of a polypeptide represents a site on the polypeptide against which an antibody may be produced and to which the antibody binds. Therefore, polypeptides comprising the amino acid sequence of a candidate gene product epitope are

useful for making antibodies to the candidate polypeptide. Preferably, an epitope comprises a sequence of at least 5, more preferably at least 10, 15, 20, 25 or 50 amino acid residues in length. Thus, polypeptides of the invention that comprises epitopes of a candidate gene product preferably contain an amino acid sequence corresponding to at least 5, at least 10, at least 15, at least 20, at least 25 or at least 50 amino acid residues of a full length candidate gene product polypeptide sequence.

Candidate gene products of the invention also include analogs and derivatives of the exemplary full length candidate gene product sequences provided in the Examples, *infra*. Analogs and derivatives of the candidate gene products of this invention have the same or homologous characteristics of the exemplary candidate gene product sequences set forth in the Examples, *infra*. Chimeric or fusion polypeptides can also be prepared in which the candidate gene product portion of the fusion polypeptide has one or more characteristics of the candidate gene product. Such fusion polypeptides therefore represent embodiments of the candidate gene product polypeptides of this invention. Such fusion polypeptides may also comprise the amino acid sequence of a marker polypeptide; for example FLAG, a histidine tag, glutathione S-transferase (GST), or the Fc portion of an IgG to name a few. Additionally, fusion polypeptides of the invention may comprise amino acid sequences that increase solubility of the polypeptide, such as a thioreductase amino acid sequence or the sequence of one or more immunoglobulin proteins (*e.g.*, IgG1 or IgG2).

Analogs or variants of a candidate polypeptide can also be made by altering encoding nucleic acid molecules, for example by substitutions, additions or deletions. Preferred analogs or variants of a candidate polypeptide are "function conservative variants" of the particular candidate polypeptide sequence specified in the Examples, *infra*. "Function-conservative variants" of a polypeptide or polynucleotide are those in which a given amino acid residue in the polypeptide, or the amino acid residue encoded by a codon of the polynucleotide, has been changed or altered without altering the overall conformation and function of the polypeptide. Such changes are expected to have little or no effect on the apparent molecular weight or isoelectric point of the polypeptide. Hence, such altered nucleic acid molecules preferably encode functionally similar molecules (*i.e.*, molecules that

perform one or more functions of a candidate polypeptide and/or have one or more of the candidate polypeptide's bioactivities).

Amino acid residues, other than ones that are specifically identified herein as being conserved, may differ among variants of a protein or polypeptide. Accordingly, the percentage of protein or amino acid sequence similarity between any two variants or analogs of a candidate polypeptide may vary. Typically, the percentage of protein or amino acid sequence similarity between variant or analog candidate polypeptides may be from 70% to 99%, as determined according to an alignment scheme such as the Cluster Method and/or the MEGALIGN or GCG alignment algorithm. Preferred variants and analogs of a candidate polypeptide are at least about 75%, and more preferably at least about 80%, 85%, 90%, 95% or 99% sequence identity as determined by a sequence comparison algorithm such as BLAST, FASTA, DNA Strider, CLUSTAL, *etc.*

Function-conservative variants of the present invention, as defined above, include not only variants of the full length candidate polypeptides of this invention (*e.g.*, variants of polypeptides comprising the particular candidate polypeptide sequences specified in the Examples, *infra*), but also include function-conservative variants of modified candidate polypeptides (*e.g.*, truncations and deletions) and of fragments (*e.g.*, corresponding to domains or epitopes) of full length candidate polypeptides.

In yet other embodiments, an analog of a candidate polypeptide is an allelic variant or mutant of a candidate polypeptide sequence provided, *e.g.*, in the Examples, *infra*. The terms allelic variant and mutant, when used herein to describe a polypeptide, refer to a polypeptide encoded by an allelic variant or mutant gene. Thus, the allelic variant and mutant candidate polypeptides of this invention are polypeptides encoded by allelic variants or mutants of a candidate nucleic acid of the present invention.

In yet other embodiments, an analog of a candidate polypeptide is a substantially homologous polypeptide from the same species (*e.g.*, allelic variants) or from another species (*e.g.*, an orthologous polypeptide). The term "homologous," in all its

grammatical forms and spelling variations, refers to the relationship between two proteins or nucleic acids that possess a "common evolutionary origin", including proteins from superfamilies (*e.g.*, the immunoglobulin superfamily) in the same species of organism as well as homologous proteins from different species of organism (for example, myosin light chain polypeptide, *etc.*; see, Reeck *et al.*, *Cell* 1987, 50:667). Such proteins (and their encoding nucleic acids) having sequence homology, as reflected by their sequence similarity, whether in terms of percent identity or by the presence of specific residues or motifs and conserved positions. Preferred homologous polypeptides of the present invention have levels of sequence similarity or identity as specified, above, for other variant and analog candidate polypeptides of the invention. Homologs and orthologs of the specific candidate polypeptides may be obtained, *e.g.*, from mammals such as humans, mice, rats, hamsters, rabbit, guinea pig, dog, cat, sheep, goat, pig, horse and cow to name a few.

In other embodiments, variants of a candidate polypeptide (including analogs, homologs, *etc.*) are polypeptides encoded by nucleic acid molecules that hybridize to the complement of a nucleic acid molecule encoding one or more of the particular candidate polypeptide sequences specified in the Examples, *infra*. A nucleic acid molecule is "hybridizable" to another nucleic acid molecule (for example cDNA, genomic DNA, or RNA) when a single stranded form of the nucleic acid molecule can anneal to the other nucleic acid molecule under appropriate conditions of temperature and solution ionic strength (see, *e.g.*, Sambrook *et al.*, *supra*). The conditions of temperature and ionic strength determine the "stringency" of the hybridization. For preliminary screening for homologous nucleic acids, low stringency hybridization conditions corresponding to a melting temperature (T_m) of about 55 °C can be used (for example, 5x SSC, 0.1% SDS, 0.25% milk and no formamide; or, alternatively, 30% formamide, 5x SSC, and 0.5% SDS). Moderate stringency hybridization conditions correspond to a higher T_m., *e.g.*, 40% formamide with 5x or 6x SSC. High stringency hybridization conditions correspond to the highest T_m, *e.g.*, 50% formamdie, 5x or 6x SSC. A 1x SSC solution is understood to be a solution containing 0.15 M NaCl and 0.015 M Na-citrate.

Hybridization requires that the two nucleic acids contain complementary sequences, although depending on the stringency of the hybridization, mismatches between bases are possible. The appropriate stringency for hybridizing nucleic acids depends on the length of the nucleic acids and the degree of complementation, variables well known in the art. The greater the degree of similarity or homology between two nucleotide sequences the greater the value of T_m for hybrids of nucleic acids having those sequences.

For hybrids of greater than 100 nucleotides in length, equations for calculating T_m have been derived (see, Sambrook *et al.*, *supra*, 9.50-9.51).

In a specific embodiment, the term "standard hybridization conditions" refers to a T_m of about 55 °C and utilizes conditions as set forth above. In a preferred embodiment, the Tm is 60 °C; in a more preferred embodiment, the Tm is 65 °C. In a specific embodiment, the term "high stringency" refers to hybridization and/or washing conditions at 68 °C in 0.2x SSC, at 42 °C in 50% formamide, 4x SSC, or under conditions that afford levels of hybridization equivalent to those observed under either of these two conditions.

In still other embodiments, variants (including analogs, homologs and orthologs) of a candidate polypeptide can be identified by isolating variants of a candidate gene, *e.g.*, using PCR with degenerate oligonucleotide primers designed on the basis of amino acid sequences of the candidate polypeptides and as described below.

Derivatives of a candidate polypeptide of the invention further include phosphorylated polypeptides, myristylated polypeptides, methylated polypeptides, and other candidate polypeptides that are chemically modified. Candidate polypeptides of the invention further include labeled variants; for example, radio-labeled with iodine or phosphorous (see, *e.g.*, EP 372707B) or other detectable molecules such as, but by no means limited to, biotin, fluorescent dyes (*e.g.*, Cy5 or Cy3), a chelating group complexed with a metal ion, a chromophore or fluorophore, a gold colloid, a particle such as a latex bead, or attached to a water soluble polymer such as poly(ethylene)-glycol (PEG). Chemical modifications of a candidate polypeptide may provide additional advantages under certain

circumstances. See, for example, U.S. Patent No. 4,179,337. For a review, see also Abuchowski *et al.*, in *Enzymes as Drugs* (J.S. Holcerberg & J. Roberts, eds. 1981) pages 367-383. A review article describing protein modification and fusion proteins is also found in Fracis, *Focus on Growth Factors* 1992, 3:4-10, Mediscript: Mountview Court, Friern Barnet Lane, London N20, OLD, UK.

Candidate Nucleic Acids:

It is understood that, for purposes of describing the present invention, the term "candidate nucleic acid" refers to a nucleic acid comprising the nucleotide sequence of a candidate gene. For convenience, candidate nucleic acids of the present invention are frequently identified here by the SEQ ID number or GenBank Accession number for their preferred nucleotide sequences or for preferred amino acid sequences that they encode. However, it is understood that, as with the candidate polypeptides, the candidate nucleic acids of this invention are not limited to those particular sequences and include homologs and variants that are well within the ordinary skill of the art.

In general, candidate nucleic acid molecule of the present invention comprises a nucleic acid sequence that encodes a candidate polypeptide as defined, *supra*, the complement of a nucleic acid sequence that encodes a candidate polypeptide, and fragments thereof. Thus, the exemplary nucleic acid sequences provided in GenBank Accession numbers specified for particular candidate genes of the Examples, *infra*, represent preferred candidate nucleic acid sequences of the present invention.

In still other embodiments, the candidate nucleic acid molecules of the invention comprise nucleotide sequences that encode one or more domains of a candidate polypeptide.

The candidate nucleic acid molecules of the invention also include nucleic acids which comprise a sequence encoding one or more fragments of a candidate polypeptide sequence.

The candidate nucleic acid molecules of the invention also include nucleic acid molecules that comprise coding sequences for modified candidate polypeptides (*e.g.*, having amino acid substitutions, deletions or truncations) and for variants (including allelic variants, analogs and homologs from the same or different species) candidate polypeptides. In preferred embodiments, such nucleic acid molecules have at least 50%, preferably at least 75% and more preferably at least 90% sequence identity to candidate polypeptide coding sequence (*e.g.*, to the coding sequence set forth in the Examples, *infra*).

In addition, candidate nucleic acid molecules of the invention include ones that hybridize to another candidate nucleic acid molecule, *e.g.*, in a Southern blot assay under defined conditions. For example, in specific embodiments a candidate nucleic acid molecule of the invention comprises a nucleotide sequence which hybridizes to a complement of a particular nucleic acid sequence, such as the coding sequence set forth in the GenBank Accession numbers for exemplary candidate genes specified in the Examples, *infra*. Alternatively, a nucleic acid molecule of the invention may hybridize, under the same defined hybridization conditions, to the complement of a fragment of a nucleotide sequence encoding a full length candidate polypeptide. Examples of preferred hybridization include those set forth above.

In other embodiments, the nucleic acid molecules of the invention comprise fragments of a full length candidate nucleic acid sequence. Such candidate nucleic acid fragments comprise a nucleotide sequence that corresponds to a sequence of at least 10 nucleotides, preferably at least 15 nucleotides and more preferably at least 20 nucleotides of a nucleotide sequence encoding a full length candidate polypeptide. In preferred embodiments, the candidate nucleic acid fragments comprise sequences of at least 10, preferably at least 15, and more preferably at least 20 nucleotides that are complementary and/or hybridize to a full length candidate nucleic acid sequence or to a fragment thereof. For hybridization with shorter nucleic acids, *i.e.*, oligonucleotides, the position of mismatches becomes more important and the length of the oligonucleotide determines its specificity (see, Sambrook *et al.*, *supra*, at 11.7-11.8). A minimum length for a hybridizable nucleic acid is preferably at

least about 10 nucleotides, more preferably at least about 15 nucleotides, and still more preferably at least about 20 nucleotides.

Nucleic acid molecules comprising such fragments are useful, for example, as oligonucleotide probes and primers (*e.g.*, PCR primers) to detect and amplify other nucleic acid molecules encoding a candidate polypeptide, including genes the encode variant candidate polypeptides. Oligonucleotide fragments of the invention may also be used, *e.g.*, as antisense nucleic acids to modulate levels of a candidate gene's expression or transcription in cells.

The nucleic acid molecules of the invention also include "chimeric" nucleic acid molecules. Such chimeric nucleic acid molecules are polynucleotides which comprise at least one candidate nucleic acid sequence (which may be any of the full length or partial candidate nucleic acid sequences described above), and also at least one non-candidate nucleic acid sequence (*i.e.*, a nucleic acid sequence not normally associated with the particular candidate gene). For example, the non-candidate nucleic acid sequence may be a heterologous regulatory sequence (for example a promoter sequence) that is derived from another gene and is not normally associated with the naturally occurring candidate gene. The non-candidate nucleic acid sequence may also be a coding sequence of another polypeptide such as FLAG, a histidine tag, glutathione S-transferase (GST), hemaglutinin, β-galactosidase, thioreductase or an immunoglobulin domain or domains (for examples, an Fc region). In preferred embodiments, a chimeric nucleic acid molecule of the invention encodes a fusion polypeptide of the invention.

Nucleic acid molecules of the invention, whether genomic DNA, cDNA or otherwise, can be isolated from any source including, for example, cDNA or genomic libraries derived from a cell or cell line from an organism that has the desired candidate gene. In the case of cDNA libraries, such libraries are preferably derived from a cell or cell line that expresses the particular candidate gene. Methods for obtaining candidate genes are well known in the art (see, *e.g.*, Sambrook *et al.*, 1989, *supra*).

The DNA may be obtained by standard procedures known in the art from cloned DNA (for example, from a DNA "library"), and preferably is obtained from a cDNA library prepared from tissues with high level expression of the protein. In one preferred embodiment, the DNA is obtained from a "subtraction" library to enrich the library for cDNAs of genes specifically expressed by a particular cell type or under certain conditions. Use of such a subtraction library may increase the likelihood of isolating cDNA for a particular gene. In still other embodiments, a library may be prepared by chemical synthesis, by cDNA cloning, or by the cloning of genomic DNA or fragments thereof purified from the desired cell (See, for example, Sambrook *et al.*, 1989, *supra*; Glover, D.M. ed., 1985, *DNA Cloning: A Practical Approach*, MRL Press, Ltd. Oxford, U.K. Vols. I and II).

In one embodiment, a cDNA library may be screened for a desired candidate nucleic acid by identifying cDNA inserts that encode a polypeptide which is homologous or substantially similar to a candidate polypeptide of particular interest. Similarly, a cDNA library may be screened for a desired candidate nucleic acid by identifying cDNA inserts having a nucleic acid sequence that is homologous or substantially similar to a particular candidate nucleic acid sequence of interest.

Clones derived from genomic DNA may contain regulatory and intron DNA regions in addition to coding regions. Clones derived from cDNA generally will not contain intron sequences. Whatever the source, the gene is preferably molecularly cloned into a suitable vector for propagation of the gene. Identification of the specific DNA fragment containing the desired candidate gene may be accomplished in a number of ways. For example, a portion of a candidate gene can be purified and labeled to prepare a labeled probe (Benton & Davis, *Science* 1977, 196:180; Grunstein & Hogness, *Proc. Natl. Acad. Sci. U.S.A.* 1975, 72:3961). Those DNA fragments with substantial homology to the probe, such as an allelic variant from another individual, will hybridize. In a specific embodiment, highest stringency hybridization conditions are used to identify a homologous candidate gene.

The genes encoding derivatives and analogs of a candidate gene of this invention can be produced by various methods known in the art. The manipulations which

result in their production can occur at the gene or protein level. For example, the cloned sequence can be modified by any of numerous strategies known in the art (Sambrook *et al.*, 1989, *supra*). The sequence can be cleaved at appropriate sites with restriction endonuclease(s), followed by further enzymatic modification if desired, isolated, and ligated *in vitro*. In the production of the gene encoding a derivative or analog of a candidate gene, care should be taken to ensure that the modified gene remains within the same translational reading frame as the candidate gene from which it is derived, uninterrupted by translational stop signals, in the gene region where the desired activity is encoded.

Additionally, a candidate gene sequence can be mutated *in vitro* or *in vivo*, to create and/or destroy translation, initiation, and/or termination sequences, or to create variations in coding regions and/or form new restriction endonuclease sites or destroy preexisting ones, to facilitate further *in vitro* modification. Modifications can also be made to introduce restriction sites and facilitate cloning the candidate gene into an expression vector. Any technique for mutagenesis known in the art can be used, including but not limited to, *in vitro* site-directed mutagenesis (Hutchinson, C., *et al.*, J. Biol. Chem. 253:6551, 1978; Zoller and Smith, DNA 3:479-488, 1984; Oliphant *et al.*, Gene 44:177, 1986; Hutchinson *et al.*, Proc. Natl. Acad. Sci. U.S.A. 83:710, 1986), use of TAB™ linkers (Pharmacia Corp., Peapack, NJ), *etc.* PCR techniques are preferred for site directed mutagenesis (see Higuchi, 1989, "Using PCR to Engineer DNA", in *PCR Technology: Principles and Applications for DNA Amplification*, H. Erlich, ed., Stockton Press, Chapter 6, pp. 61-70).

The identified and isolated gene can then be inserted into an appropriate cloning vector. A large number of vector-host systems known in the art may be used. Possible vectors include, but are not limited to, plasmids or modified viruses, but the vector system must be compatible with the host cell used. Examples of vectors include, but are not limited to, *E. coli*, bacteriophages such as lambda derivatives, or plasmids such as pBR322 derivatives or pUC plasmid derivatives, *e.g.*, pGEX vectors, pmal-c, pFLAG, pKK plasmids (Clonetech, Palo Alto, CA), pET plasmids (Novagen, Inc., Madison, WI), pRSET or pREP plasmids, pcDNA (Invitrogen, Carlsbad, CA), or pMAL plasmids (New England Biolabs, Beverly, MA), *etc.* The insertion into a cloning vector can, for example, be accomplished by

ligating the DNA fragment into a cloning vector which has complementary cohesive termini. However, if the complementary restriction sites used to fragment the DNA are not present in the cloning vector, the ends of the DNA molecules may be enzymatically modified. Alternatively, any site desired may be produced by ligating nucleotide sequences (linkers) onto the DNA termini. These ligated linkers may comprise specific chemically synthesized oligonucleotides encoding restriction endonuclease recognition sequences.

Recombinant molecules can be introduced into host cells via transformation, transfection, infection, electroporation, etc., so that many copies of the gene sequence are generated. Preferably, the cloned gene is contained on a shuttle vector plasmid, which provides for expansion in a cloning cell, *e.g.*, *E. coli*, and facile purification for subsequent insertion into an appropriate expression cell line, if such is desired. For example, a shuttle vector, which is a vector that can replicate in more than one type of organism, can be prepared for replication in both *E. coli* and *Saccharomyces cerevisiae* by linking sequences from an *E. coli* plasmid with sequences from the yeast 2m plasmid.

It is understood that candidate nucleic acids of the invention may be either DNA or RNA and may be single-, double- or even triple-stranded (*e.g.*, a triple-helix of candidate single-stranded candidate nucleic acids and/or their complement(s)). Candidate nucleic acids of the invention include genomic DNA, cDNA, RNA, mRNA, cRNA, *etc.*; as well as synthetic and genetically manipulated polynucleotides and both sense and antisense polynucleotides. Such synthetic polynucleotides include, for example, "protein nucleic acids" (PNA) formed by conjugating nucleotide bases to an amino acid backbone. Other exemplary synthetic nucleic acids include nucleic acids containing modified bases, such as thio-uracil, thio-guanine and fluoro-uracil. For convenience, the exemplary nucleotide sequences provided in this description are provided as sequences of DNA. However, it is understood that identical sequences of other types of nucleic acids (for example, RNA) may also be used and are equivalent. Thus, for example, where the particular nucleotide sequences in this description specify a thymine (T) at some position, it is understood that a uracil (U) may be substituted at that position and is a functional equivalent.

The polynucleotides of this invention may be flanked by natural regulatory sequences, or they may be associated with heterologous sequences such as promoters, enhancers, response elements, signal sequences, polyadenylation sequences, introns, 5' and 3'-non-coding regions and the like. The term "heterologous", in this context, refers to a combination of elements (*e.g.*, sequences) that are not naturally occurring. Hence, a candidate nucleic acid of this invention may have sequences, such as a promoter *etc.*, that are not normally associated with the candidate gene.

Nucleic acids of the invention may also be modified by any means known in the art. Non-limiting examples of such modifications include methylation, "caps", substitution of one or more of the naturally occurring nucleotides with an analog, and internucleotide modifications such as, for example, those with uncharged linkages (*e.g.*, methyl phosphonates, phosphotriesters, phosphoroamidates, carbamates, *etc.*) and with charged linkages (*e.g.*, phosphorothioates, phosphorodithioates, *etc.*). Nucleic acids of the invention may contain one or more additional covalently linked moieties such as proteins (*e.g.*, nucleases, toxins, antibodies, signal peptides, poly-L-lysine, *etc.*), intercalators (*e.g.*, acridine, psoralen, *etc.*), chelators (*e.g.*, metals, radioactive metals, iron, oxidative metals, *etc.*) and alkylators to name a few. The polynucleotides may be derivatized by formation of a methyl or ethyl phosphotriester or an alkyl phosphoramidite linkage. Furthermore, the polynucleotides herein may also be modified with a label capable of providing a detectable signal, either directly or indirectly. Exemplary labels include radioisotopes, fluorescent molecules, biotin and the like.

Expression of Candidate Polypeptides and Nucleic Acids:

A nucleotide sequence coding for candidate polypeptides , including chimeric proteins, antigenic fragments, derivatives or analogs thereof may be inserted into an appropriate expression vector, *i.e.*, a vector which contains the necessary elements for the transcription and translation of the inserted protein-coding sequence. Thus, a nucleic acid encoding a candidate polypeptide of the invention can be operationally associated with a promoter in an expression vector of the invention. Both cDNA and genomic sequences can

be cloned and expressed under control of such regulatory sequences. Such vectors can be used to express functional or functionally inactivated candidate polypeptides.

The necessary transcriptional and translational signals can be provided on a recombinant expression vector.

Potential host-vector systems include but are not limited to mammalian or other vertebrate cell systems transfected with expression plasmids or infected with virus (*e.g.*, vaccinia virus, adenovirus, adeno-associated virus, herpes virus, etc.); insect cell systems infected with virus (*e.g.*, baculovirus); microorganisms such as yeast containing yeast vectors; or bacteria transformed with bacteriophage, DNA, plasmid DNA, or cosmid DNA. The expression elements of vectors vary in their strengths and specificities. Depending on the host-vector system utilized, any one of a number of suitable transcription and translation elements may be used.

Expression of a candidate protein may be controlled by any promoter/enhancer element known in the art, but these regulatory elements must be functional in the host selected for expression. Promoters which may be used to control MIP–3α gene expression include, but are not limited to, cytomegalovirus (CMV) promoter (U.S. Patent Nos. 5,385,839 and 5,168,062), the SV40 early promoter region (Benoist and Chambon, *Nature* 1981, 290:304-310), the promoter contained in the 3' long terminal repeat of Rous sarcoma virus (Yamamoto, *et al.*, *Cell* 1980, 22:787-797), the herpes thymidine kinase promoter (Wagner *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 1981, 78:1441-1445), the regulatory sequences of the metallothionein gene (Brinster *et al.*, *Nature* 1982, 296:39-42); prokaryotic expression vectors such as the b-lactamase promoter (Villa-Komaroff, *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 1978, 75:3727-3731), or the *tac* promoter (DeBoer, *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 1983, 80:21-25, 1983); see also "Useful proteins from recombinant bacteria" in *Scientific American* 1980, 242:74-94. Still other useful promoter elements which may be used include promoter elements from yeast or other fungi such as the Gal 4 promoter, the ADC (alcohol dehydrogenase) promoter, PGK (phosphoglycerol kinase) promoter, alkaline phosphatase promoter; and transcriptional control regions that exhibit hematopoietic tissue

specificity, in particular: beta-globin gene control region which is active in myeloid cells (Mogram *et al.*, *Nature* 1985, 315:338-340; Kollias *et al.*, *Cell* 1986, 46:89-94), hematopoietic stem cell differentiation factor promoters, erythropoietin receptor promoter (Maouche *et al.*, *Blood* 1991, 15:2557), *etc.*

In another embodiment, the invention provides methods for expressing candidate polypeptides by using a non-endogenous promoter to control expression of endogenous candidate genes within a cell. An endogenous candidate gene within a cell is a candidate gene of the present invention which is ordinarily (*i.e.*, naturally) found in the genome of that cell. A non-endogenous promoter, however, is a promoter or other nucleotide sequence that may be used to control expression of a gene but is not ordinarily or naturally associated with the endogenous candidate gene. As an example, methods of homologous recombination may be employed (preferably using non-protein encoding nucleic acid sequences of the invention) to insert an amplifiable gene or other regulatory sequence in the proximity of an endogenous candidate gene. The inserted sequence may then be used, *e.g.*, to provide for higher levels of the candidate gene's expression than normally occurs in that cell, or to overcome one or more mutations in the endogenous candidate gene's regulatory sequences which prevent normal levels of gene expression. Such methods of homologous recombination are well known in the art. See, for example, International Patent Publication No. WO 91/06666, published May 16, 1991 by Skoultchi; International Patent Publication No. WO 91/099555, published July 11, 1991 by Chappel; and International Patent Publication No. WO 90/14092, published November 29, 1990 by Kucherlapati and Campbell.

Soluble forms of the protein can be obtained by collecting culture fluid, or solubilizing inclusion bodies, *e.g.*, by treatment with detergent, and if desired sonication or other mechanical processes, as described above. The solubilized or soluble protein can be isolated using various techniques, such as polyacrylamide gel electrophoresis (PAGE), isoelectric focusing, 2-dimensional gel electrophoresis, chromatography (*e.g.*, ion exchange, affinity, immunoaffinity, and sizing column chromatography), centrifugation, differential solubility, immunoprecipitation, or by any other standard technique for the purification of proteins.

Preferred vectors are viral vectors, such as lentiviruses, retroviruses, herpes viruses, adenoviruses, adeno-associated viruses, vaccinia virus, baculovirus, and other recombinant viruses with desirable cellular tropism. Thus, a gene encoding a functional or mutant candidate protein or polypeptide domain fragment thereof can be introduced *in vivo*, *ex vivo*, or *in vitro* using a viral vector or through direct introduction of DNA. Expression in targeted tissues can be effected by targeting the transgenic vector to specific cells, such as with a viral vector or a receptor ligand, or by using a tissue-specific promoter, or both.

Antibodies to Candidate Gene Products:

Antibodies to candidate gene products of the present invention are useful, *inter alia*, for diagnostic and therapeutic methods, as set forth below. According to the invention, candidate polypeptides produced, *e.g.*, recombinantly or by chemical synthesis, and fragments or other derivatives or analogs thereof, including fusion proteins, may be used as an immunogen to generate antibodies that recognize these polypeptides. Such antibodies include but are not limited to polyclonal, monoclonal, chimeric, single chain, Fab fragments, and an Fab expression library. Such an antibody is preferably specific for (*i.e.*, specifically binds to) a human candidate polypeptide of the present invention. However, the antibody may, alternatively, be specific for an ortholog from some other species of organism, preferably another species of mammal such as mouse, rat or hamster, to name a few. The antibody may recognize wild-type, mutant or both forms of the candidate polypeptide.

Various procedures known in the art may be used for the production of polyclonal antibodies. For the production of polyclonal antibodies, various host animals can be immunized by injection with the desired candidate polypeptide, or derivatives (*e.g.*, fragments or fusion proteins) thereof, including but not limited to rabbits, mice, rats, sheep, goats, etc. In one embodiment, the candidate polypeptide or fragment thereof can be conjugated to an immunogenic carrier, *e.g.*, bovine serum albumin (BSA) or keyhole limpet hemocyanin (KLH). Various adjuvants may be used to increase the immunological response, depending on the host species, including but not limited to Freund's (complete and incomplete), mineral gels such as aluminum hydroxide, surface active substances such as

lysolecithin, pluronic polyols, polyanions, peptides, oil emulsions, keyhole limpet hemocyanins, dinitrophenol, and potentially useful human adjuvants such as BCG (*bacille Calmette-Guerin*) and *Corynebacterium parvum*.

For preparation of monoclonal antibodies directed toward the candidate polypeptides, or fragment, analogs, or derivatives thereof, any technique that provides for the production of antibody molecules by continuous cell lines in culture may be used. These include but are not limited to the hybridoma technique originally developed by Kohler and Milstein (*Nature* 1975, 256:495-497), as well as the trioma technique, the human B-cell hybridoma technique (Kozbor *et al.*, *Immunology Today* 1983, 4:72; Cote *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 1983, 80:2026-2030), and the EBV-hybridoma technique to produce human monoclonal antibodies (Cole *et al.*, in *Monoclonal Antibodies and Cancer Therapy*, Alan R. Liss, Inc., 1985, pp. 77-96). In an additional embodiment of the invention, monoclonal antibodies can be produced in germ-free animals (International Patent Publication No. WO 89/12690). In fact, according to the invention, techniques developed for the production of "chimeric antibodies" (Morrison *et al.*, *J. Bacteriol.* 1984, 159:870; Neuberger *et al.*, *Nature* 1984, 312:604-608; Takeda *et al.*, *Nature* 1985, 314:452-454) may also be used. Briefly, such techniques comprise splicing the genes from an antibody molecule from a first species of organism (*e.g.*, a mouse) that is specific for a candidate polypeptide together with genes from an antibody molecule of appropriate biological activity derived from a second species of organism (*e.g.*, from a human). Such chimeric antibodies are within the scope of this invention.

Antibody fragments which contain the idiotype of the antibody molecule can be generated by known techniques. For example, such fragments include but are not limited to: the $F(ab')_2$ fragment which can be produced by pepsin digestion of the antibody molecule; the Fab' fragments which can be generated by reducing the disulfide bridges of the $F(ab')_2$ fragment, and the Fab fragments which can be generated by treating the antibody molecule with papain and a reducing agent.

According to the invention, techniques described for the production of single chain antibodies (U.S. Patent Nos. 5,476,786, 5,132,405, and 4,946,778) can be adapted to produce specific single chain antibodies that specifically bind to a particular candidate polypeptide. An additional embodiment of the invention utilizes the techniques described for the construction of Fab expression libraries (Huse *et al.*, *Science* 1989, 246:1275-1281) to allow rapid and easy identification of monoclonal Fab fragments with the desired specificity for a candidate polypeptide, or for its derivatives, or analogs.

In the production and use of antibodies, screening for or testing with the desired antibody can be accomplished by techniques known in the art, *e.g.*, radioimmunoassay, ELISA (enzyme-linked immunosorbant assay), "sandwich" immunoassays, immunoradiometric assays, gel diffusion precipitin reactions, immunodiffusion assays, *in situ* immunoassays (using colloidal gold, enzyme or radioisotope labels, for example), Western blots, precipitation reactions, agglutination assays (*e.g.*, gel agglutination assays, hemagglutination assays), complement fixation assays, immunofluorescence assays, protein A assays; and immunoelectrophoresis assays, etc. In one embodiment, antibody binding is detected by detecting a label on the primary antibody. In another embodiment, the primary antibody is detected by detecting binding of a secondary antibody or reagent to the primary antibody. In a further embodiment, the secondary antibody is labeled. Many means are known in the art for detecting binding in an immunoassay and are within the scope of the present invention.

The foregoing antibodies can be used in methods known in the art relating to the localization and activity of a candidate polypeptide of interest, *e.g.*, for Western blotting, imaging candidate polypeptides *in situ*, measuring levels thereof in appropriate physiological samples, *etc.* using any of the detection techniques mentioned above or known in the art. Such antibodies can also be used in assays for ligand binding, *e.g.*, as described in US Patent No. 5,679,582. Antibody binding generally occurs most readily under physiological conditions, *e.g.*, pH of between about 7 and 8, and physiological ionic strength. The presence of a carrier protein in the buffer solutions stabilizes the assays. While there is some tolerance of perturbation of optimal conditions, *e.g.*, increasing or decreasing ionic strength,

temperature, or pH, or adding detergents or chaotropic salts, such perturbations generally decrease binding stability.

In still other embodiments, antibodies may also be used to isolate cells which express a candidate polypeptide of interest (for example, OA chondrocyte cells) by panning or related immunoadsorption techniques.

In a specific embodiment, antibodies that agonize or antagonize the activity of a candidate polypeptide can be generated. In particular, intracellular single chain Fv antibodies can be used to regulate (inhibit) MIP–3α activity (Marasco *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 1993, 90:7884-7893; Chen., *Mol. Med. Today* 1997, 3:160-167; Spitz *et al.*, *Anticancer Res.* 1996, 16:3415-22; Indolfi *et al.*, *Nat. Med.* 1996, 2:634-635; Kijma *et al.*, *Pharmacol. Ther.* 1995, 68:247-267). Such antibodies can be tested using the assays described *infra* for identifying ligands.

Applications and Uses:

Described herein are various applications and uses for candidate genes and gene products that are identified in screening methods of the present invention. These include, *inter alia*, applications and uses for the candidate nucleic acids and polypeptides described above, including the particular candidate nucleic acids and polypeptides provided in the examples as well as fragments, analogs, homologs and other variants thereof.

The candidate genes and gene products that are identified in screening assays of this invention include ones that are expressed at elevated levels in cells from patients with OA compared to healthy subjects. Other candidate genes and gene products of the invention induce one or more features of an OA phenotype when they are expressed in cells. Hence, candidate genes and/or gene products may be used as tissue-specific markers to detect and/or identify OA cells or tissue, including OA chondrocyte cells and cartilage. Candidate nucleic acids and polypeptides of the invention can therefore be used in methods for detecting OA, *e.g.*, in diagnostic and prognostic applications, by using one or more candidate genes or gene

products to detect expression in a sample such as a cell or tissue sample from an individual (obtained, *e.g.*, from a biopsy).

In addition, candidate genes and gene products of the invention can serve as drug targets for the development of therapeutics to treat individuals suffering from OA. Methods are provided that use candidate nucleic acids and polypeptides of the invention to screen for compounds that can be used to treat or prevent cartilage degradation, as well as for the treatment or prevention of conditions such as OA. Such screening methods may, for example, identify compounds that modulate or interfere with binding of a candidate gene or gene product to its ligand or receptor. In other embodiments, drug screening methods of the invention may identify compounds that modulate downstream signaling events from a candidate or gene or gene product, or they may identify compounds that interfere with upstream signaling event that activate a candidate gene or gene product. In still other embodiments, drug screening assays of the invention may identify compounds that inhibit the expression and/or activity of either a candidate gene or its gene product.

Drug screening assays. Using screening assays such as those described below, it is possible to identify compounds that bind to or otherwise interact with candidate genes of the present invention and/or their gene products, including intracellular compounds (for example, proteins or portions of proteins), natural and synthetic ligands or receptors, compounds that interfere with the interaction of a candidate gene product (for example, compounds that interfere with specific binding of a candidate gene product to its receptor or ligand), and compounds that modulate the activity of a candidate gene (for example, by modulating the level of the candidate gene's expression) or the activity (for example, the bioactivity) of a candidate gene product.

The screening assays of this invention may therefore be used to identify compounds that specifically bind to a candidate gene or gene product to modulate its expression. For example, the screening assays described here may be used to identify compounds that bind to a promoter or other regulatory sequence of a candidate gene, and so may modulate the level of that candidate gene's expression (see, for example, Platt, *J. Biol.*

Chem. 1994, 269:28558-28562). The screening assays may also be used to identify compounds that bind to and thereby stabilize a candidate nucleic acid or polypeptide. In addition, these screening assays may be used to identify compounds that inhibit or modulate such binding interactions and which are therefore useful, *e.g.*, as agonists or antagonists for the candidate gene product's binding to a specific transcription factor or enhancer, or for the candidate gene product's binding to a stabilizer. Compounds identified in these or similar screening assays may therefore be used to treat diseases and disorders that are associated with the candidate gene's abnormal expression and/or activity, associated with , but not limited to, OA.

Classes of compounds that may be identified by such screening assays include, but are not limited to, small molecules (*e.g.*, organic or inorganic molecules which are less than about 2 kDa in molecular weight, are more preferably less than about 1 kDa in molecular weight, and/or are able to cross the blood-brain barrier or gain entry into an appropriate cell and affect expression of either a candidate gene or of some gene involved in the candidate gene's regulatory pathway) as well as macromolecules (*e.g.*, molecules greater than about 2 kDa in molecular weight). Compounds identified by these screening assays may also include nucleic acids, peptides and polypeptides. Examples of such compounds (including peptides) include but are not limited to: soluble peptides; fusion peptide members of combinatorial libraries (such as ones described by Lam *et al.*, *Nature* 1991, 354:82-84; and by Houghten *et al.*, *Nature* 1991, 354:84-86); members of libraries derived by combinatorial chemistry, such as molecular libraries of D- and/or L-configuration amino acids; phosphopeptides, such as members of random or partially degenerate, directed phosphopeptide libraries (see, *e.g.*, Songyang *et al.*, *Cell* 1993, 72:767-778); antibodies, including but not limited to polyclonal, monoclonal, humanized, anti-idiotypic, chimeric or single chain antibodies; antibody fragments, including but not limited to Fab, $F(ab')_2$, Fab expression library fragments, and epitope-binding fragments thereof. Nucleic acids used in these screening assays may be DNA or RNA, or synthetic nucleic acids. Particular examples include, but are by no means limited to, antisense nucleic acids and ribozymes, as well as double-stranded and triple helix nucleic acid molecules.

Assays for binding compounds. *In vitro* systems can be readily designed to identify compounds capable of binding to a candidate gene product of the present invention. Such compounds can be useful, for example, in modulating the expression, stability or activity of a wild-type candidate gene product or, alternatively, to modulate the expression, stability or activity of a mutant or other variant candidate gene product.

Generally, such screening assays involve preparation of a reactive mixture comprising the candidate gene product of interest and a test compound under conditions and for a time sufficient to allow the two compounds to interact (*e.g.*, bind), thereby forming a complex that may be detected. The assays may be conducted in any of a variety of different ways. For example, one embodiment comprises anchoring a candidate polypeptide or a test compound onto a solid phase and detecting complexes of the candidate polypeptide and the test compound that are on the solid phase at the end of the reaction and after removing (*e.g.*, by washing) unbound compounds. For example, in one preferred embodiment of such a method, a candidate gene product may be anchored onto a solid surface and a labeled compound (*e.g.*, labeled according to any of the methods described *supra*) is contacted to the surface. After incubating the test compound for a sufficient time and under sufficient conditions that a complex may form between the candidate gene product and the test compound, unbound molecules of the test compound are removed from the surface (*e.g.*, by washing) and labeled molecules which remain are detected.

In another, alternative embodiment, molecules of one or more different test compounds are attached to the solid phase and molecules of a labeled candidate polypeptide may be contacted thereto. In such embodiments, the molecules of different test compounds are preferably attached to the solid phase at a particular location on the solid phase so that test compounds that bind to the candidate polypeptide may be identified by determining the location of the bound candidate polypeptides on the solid phase or surface.

Assays for compounds that interact with a candidate gene or gene product. Any of a variety of known methods for detecting protein-protein interactions may also be used to detect and/or identify proteins that interact with a candidate gene product of the

invention. For example, co-immunoprecipitation, cross-linking and co-purification through gradients or chromatographic columns as well as other techniques known in the art may be employed. Proteins which may be identified using such assays include, but are not limited to, extracellular proteins, such as receptors and ligands for candidate genes and/or their gene products, as well as intracellular proteins such as signal transducing proteins.

Compounds, including other cellular proteins and nucleic acids, that interact with a candidate gene or gene product may themselves be used in the methods of this invention, *e.g.*, to modulate activity of the candidate gene or gene product and to treat or prevent cartilage degradation. Alternatively, such interacting compounds may, themselves, be used in the screening assays of this invention to identify other compounds that could, in turn, be used to treat or prevent cartilage degradation.

As an example, and not by way of limitation, an expression cloning assay may be used to identify receptors and other proteins that specifically interact with a candidate gene product of interest. In such assays, a cDNA expression library may be generated from any cell line that expresses such a receptor. Clones from such an expression library may then be transfected or infected into cells that do not normally express a receptor for the candidate gene product. Cells that are transfected with a clone that encodes a receptor which specifically binds to the candidate gene product may then express this receptor, and can be identified and isolated using standard techniques such as FACS or using magnetic beads that have the candidate polypeptide (for example, an Fc-fusion of the candidate polypeptide) attached thereto.

Alternatively, receptors and/or ligands that specifically bind to a candidate gene product may be isolated from a cell line using immunoprecipitation techniques that are well known in the art.

Receptors and/or ligands for a candidate gene product may also be isolated using any of the screening assays discussed, *supra* for identifying binding compounds. For example, an Fc-fusion polypeptide of a candidate gene product may be bound or otherwise

attached to a solid surface, and a labeled compound (*e.g.*, a candidate receptor or ligand) may be contacted to the surface for a sufficient time and under conditions that permit formation of a complex between the fusion polypeptide and the test compound. Unbound molecules of the test compound can then be removed from the surface (*e.g.*, by washing), and labeled compounds that remain bound can be detected.

Once so isolated, standard techniques may be used to identify any protein detected in such assays. For example, at least a portion of the amino acid sequence of a protein that interacts with a candidate gene product can be ascertained using techniques well known in the art, such as the Edman degradation technique (see, *e.g.*, Creighton, 1983, Proteins: Structures and Molecular Principles, W.H. Freeman&Co., New York, pages 34-49).

Once such proteins have been identified, their amino acid sequence may be used as a guide for the generation of oligonucleotide mixtures to screen for gene sequences encoding such proteins; *e.g.*, using standard hybridization or PCR techniques described *supra*. See, for example, Ausubel *supra*; and PCR Protocols: A Guide to Methods and Applications, Innis *et al.*, eds., Academic Press, Inc., New York (1990) for descriptions of techniques for the generation of such oligonucleotide mixtures and their use in screening assays.

Other methods are known in the art which result in the simultaneous identification of genes that encode a protein that interacts with a candidate gene or gene product. For example, expression libraries may be probed with a labeled candidate polypeptide.

As another example and not by way of limitation, a two-hybrid system may be used to detect protein interactions with a candidate gene product *in vivo*. Briefly, utilizing such a system, plasmids may be constructed which encode two hybrid proteins, one of which preferably comprises of the DNA-binding domain of a transcription activator protein fused to a candidate gene product. The other hybrid protein preferably comprises an activation

domain of the transcription activator protein used in the first hybrid, fused to an unknown protein that is encoded by a cDNA recombined into the plasmid library as part of a cDNA library. Both the DNA-binding domain fusion plasmid and the cDNA library may be co-transformed into a strain of *Saccharomyces cerevisiae* or other suitable organism which contains a reporter gene (for example, HBS, lacZ, HIS3 or GFP). Preferably, the regulatory region of this reporter gene comprises a binding site for the transcription activator moiety of the two hybrid proteins. In such a two-hybrid system, the presence of either of the two hybrid proteins alone cannot activate transcription of the reporter gene. Specifically, the DNA-binding domain hybrid protein cannot activate transcription because it cannot localize to the necessary activation function. Likewise, the activation domain hybrid protein cannot activate transcription because it cannot localize to the DNA binding site on the reporter gene. However, interaction between the two hybrid proteins, reconstitutes that functional transcription activator protein and results in expression of the reporter gene. Thus, in a two-hybrid system such as the one described here in detail, an interaction between a candidate polypeptide (*i.e.*, the candidate polypeptide fused to the transcription activator's DNA binding domain) and a test polypeptide (*i.e.*, a protein fused to the transcription activator's DNA binding domain) may be detected by simply detecting expression of a gene product of the reporter gene.

cDNA libraries for screening in such two-hybrid and other assays may be made according to any suitable technique known in the art. As a particular and non-limiting example, cDNA fragments may be inserted into a vector so that they are translationally fused to the transcriptional activation domain of GAL4, and co-transformed along with a "bait" GAL4 fusion plasmid (encoding a GAL4-fusion of a candidate gene product) into a strain of *Saccharomyces cerevisiae* or other suitable organism that contains a HIS3 gene driven by a promoter that contains a GAL4 activation sequence. A protein from this cDNA library, fused to the GAL4 transcriptional activation domain, which interacts with the candidate polypeptide moiety of the GAL4-fusion will reconstitute and active GAL4 protein, and can thereby drive expression of the HIS3 gene. Colonies that express the HIS3 gene may be detected by their growth on petri dishes containing semi-solid agar based media lacking

histidine. The cDNA may then be purified from these strains, sequenced and used to identify the encoded protein which interacts with the candidate polypeptide.

Once compounds have been identified which bind to a candidate gene or gene product of the invention, the screening methods described in these methods may also be used to identify other compounds (*e.g.*, small molecules, peptides and proteins) which bind to these binding compounds. Such compounds may also be useful for modulating bioactivities associated with a candidate gene and its gene product, for example by binding to a natural receptor, ligand or other binding partner and preventing its interaction with the candidate gene product. For instance, these compounds could be tested for their ability to inhibit the binding of an Fc-fusion of the candidate gene product to cell lines which express a specific receptor for the candidate gene product.

Assays for compounds that interfere with a candidate gene/protein ligand interaction. As noted *supra*, a candidate gene product of the invention may interact with one or more molecules (*e.g.*, with a specific receptor or ligand) *in vivo* or *in vitro*. Compounds that disrupt or otherwise interfere with this binding interaction are therefore useful in modulating biological activity or activities that are associated with the candidate gene product, including for example, cartilage degradation. Such compounds may therefore be useful, e.g., to treat disorders such as OA that are associated with abnormal levels of a candidate gene or gene product's expression and/or activity.

Such compounds include, but are not limit to, compounds identified according to the screening assays described *supra*, for identifying compounds that bind to a candidate gene product, including any of the numerous exemplary classes of compounds described therein.

In general, assays for identifying compounds that interfere with the interaction between a candidate gene product and a binding partner (*e.g.*, a receptor or ligand) involve preparing a test reaction mixture that contains the candidate gene product and its binding partner under conditions and for a time sufficient for the candidate gene product and its

binding partner to bind and form a complex. In order to test a compound for inhibitory activity (*i.e.*, for the ability to inhibit formation of the binding complex or to disrupt the binding complex once formed), the test compound preferably is also present in the test reaction mixture. In one exemplary embodiment, the test compound may be initially included in the test reaction mixture with the candidate gene product and its binding partner. Alternatively, however, the test compound may be added to the test reaction mixture at a later time, subsequent to the addition of the candidate gene product and its binding partner. In preferred embodiments, one or more control reaction mixtures, which do not contain the test compound, may also be prepared. Typically, a control reaction mixture will contain the same candidate gene product and binding partner that are in the test reaction mixture, but will not contain a test compound. A control reaction mixture may also contain a placebo, not present in the test reaction mixture, in place of the test compound. The formation of a complex between the candidate gene product and the binding partner may then be detected in the reaction mixture. The formation of such a complex in the absence of the test compound (*e.g.*, in a control reaction mixture) but not in the presence of the test compound, indicates that the test compound is one which interferes with or modulates the interaction of the candidate polypeptide and its binding partner.

Such assays for compounds that modulate the interaction of a candidate gene product and a binding partner may be conducted in a heterogeneous format or, alternatively, in a homogeneous format. Heterogeneous assays typically involve anchoring either a candidate gene product or a binding partner onto a solid phase and detecting compounds anchored to the solid phase at the end of the reaction. Thus, such assays are similar to the solid phase assays described *supra* for detecting and/or identifying candidate nucleic acids and gene products and for detecting or identifying binding partners. Indeed, those skilled in the art will recognize that many of the principles and techniques described above for those assays may be modified and applied without undue experimentation in the solid phase assays described here, for identifying compounds that modulate interaction(s) between a candidate gene product and a binding partner.

Regardless of the particular assay used, the order to which reactants are added to a reaction mixture may be varied; for example, to identify compounds that interfere with the interaction of a candidate gene product with a binding partner by competition, or to identify compounds that disrupt a preformed binding complex. Compounds that interfere with the interaction of a candidate gene product with a binding partner by competition may be identified by conducting the reaction in the presence of a test compound. Specifically, in such assays a test compound may be added to the reaction mixture prior to or simultaneously with the candidate gene product and the binding partner. Test compounds that disrupt preformed complexes of a candidate gene product and a binding partner may be tested by adding the test compound to a reaction mixture after complexes have been formed.

The screening assays described herein may also be practiced using peptides or polypeptides that correspond to portions of a full length candidate polypeptide or protein, or with fusion proteins comprising such peptide or polypeptide sequences. For example, screening assays for identifying compounds the modulate interactions of a candidate polypeptide with a binding partner may be practiced using peptides or polypeptides corresponding to particular regions or domains of a full length candidate polypeptide that bind to a binding partner (*e.g.*, receptor "binding sites").

A variety of methods are known in the art that may be used to identify specific binding sites of a candidate polypeptide. For example, binding sites may be identified by mutating a candidate gene and screening for disruptions of binding as described above. A gene encoding the binding partner may also be mutated in such assays to identify mutations that compensate for disruptions from the mutation to the candidate gene. Sequence analysis of these mutations can then reveal mutations that correspond to the binding region of the two proteins.

In an alternative embodiment, a protein (*e.g.*, a candidate protein or a protein binding partner to a candidate protein) may be anchored to a solid surface or support using the methods described hereinabove. Another labeled protein which binds to the protein anchored to the solid surface may be treated with a proteolytic enzyme, and its fragments

may be allowed to interact with the protein attached to the solid surface, according to the methods of the binding assays described *supra*. After washing, short, labeled peptide fragments of the treated protein may remain associated with the anchored protein. These peptides can be isolated and the region of the full length protein from which they are derived may be identified by the amino acid sequence.

In still other embodiments, compounds that interfere with interactions between a candidate polypeptide and a receptor or ligand may also be identified by screening for compounds that modulate binding of the candidate polypeptide (for example, an Fc-fusion construct of the candidate polypeptide) to cells that express a specific receptor thereto.

Diagnostic and Prognostic Applications:

A variety of methods can be employed for diagnostic and prognostic methods using reagents such as the candidate nucleic acids and polypeptides described *supra* as well as antibodies directed against such candidate nucleic acids and polypeptides. For example, using the methods described here it is possible to detect expression of a candidate nucleic acid or protein in a biological sample from an individual, such as in cells or tissues in a sample (*e.g.*, from a biopsy) obtained or derived from an individual subject or patient. As explained above, candidate nucleic acids and polypeptides identified in screening assays of this invention induce one or more characteristics associated with OA when they are expressed in cells. Hence, the expression of such candidate nucleic acids and/or polypeptides at elevated levels in cells is an indication of OA or a related disorder.

Using the methods described here (as well as other methods known in the art) a skilled artisan may detect elevated levels of a candidate nucleic acid or polypeptide in a sample of cells or tissue from an individual, and may thereby detect and/or identify cells or tissue in that sample as being symptomatic of OA. In certain preferred embodiments the particular type of tissue identified in such methods is cartilage tissue. By using such methods to detect such cells or tissue in an individual, a skilled user may thereby diagnose the presence of OA in that individual.

In preferred embodiments the methods described herein are performed using pre-packaged diagnostic kits. Such kits may comprise at least one specific candidate nucleic acid or a candidate gene product specific antibody reagent. For example, said diagnostic kit may be used for detecting mRNA levels or protein levels of a candidate gene or gene product selected from the group consisting of those disclosed in Table V and Table VI, said kit comprising: (a) a polynucleotide of said candidate gene or a fragment thereof; (b) a nucleotide sequence complementary to that of (a); (c) an expression product of said candidate gene , or a fragment thereof; or (d) an antibody to said expression product and wherein components (a), (b), (c) or (d) may comprise a substantial component.

In preferred embodiments, a kit will also contain instructions for its use, *e.g.*, to detect diseased cells or tissues, or to diagnose a disorder (such as OA) associated with abnormal expression of a candidate gene or gene product. In preferred embodiments, such instructions may be packaged directly with the kit. In other embodiments, however, instructions may be provided separately. For example, the invention provides embodiments of kits where instructions for using the kit may be downloaded, *e.g.*, from the internet. A kit of the invention may also comprise, preferably in separate containers, suitable buffers and other solutions to use the reagents (*e.g.*, nucleic acid or antibody specific for a candidate gene or gene product) to detect the candidate gene or gene product. The kit and any reagent(s) contained therein may be used, for example, in a clinical setting, to diagnose patients exhibiting or suspected of having OA.

A sample comprising a cell of any cell type or tissue of any tissue type in which a candidate gene is expressed may also be used in such diagnostic methods, *e.g.*, for detection of candidate gene expression or of candidate gene products (such as candidate polypeptides), as well as for identifying cells, *e.g.* chondrocytes, that express a candidate gene or a candidate gene product. Thus, in one embodiment, the methods described herein may be performed *in situ*, *e.g.*, using cells or tissues obtained from an individual such as in a biopsy. Such methods may be useful, for example, in surgical procedures where it is desirable to identify arthritic tissue without removing benign, healthy tissue.

The methods described herein are not limited to diagnostic applications, but may also be used in prognostic applications, *e.g.*, to monitor the progression of a disease (such as OA) that is associated with abnormal expression of a candidate gene or gene product, or to monitor a therapy thereto. Accordingly, prognostic methods of the invention may comprise, in one exemplary embodiment, monitoring candidate nucleic acid or polypeptide levels in an individual during the course of a treatment or therapy (for example, a drug treatment or exercise regimen) for OA. Similarly, the methods of the invention may also be used to detect and identify diseased cells and tissue (e.g. cells overexpressing one or more candidate genes of gene products compared to non OA cells or tissue) during the course of a therapy. In such embodiments, decreasing numbers of diseased cells is generally indicative of an effective treatment. The methods of the invention may further be used, *e.g.*, to screen candidate drugs or compounds and identify ones that may be effective, *e.g.*, as anti-OA drugs. Such methods may be performed *in vivo* (*e.g.*, using an animal model) or *in vitro* (for example, in a cell culture assay). In one embodiment such methods may comprise contacting a candidate compound to a cell and identifying whether expression of a candidate gene or gene product by the cell has been inhibited. In another embodiment, a compound may be contacted to a cell or administered to an organism, and extracellular levels of candidate nucleic acid or polypeptide may be measured (for example, in cell culture media for cell culture assays, or in blood or other body fluid in an animal model assay).

Detection of candidate nucleic acids. The diagnostic and prognostic methods of the invention include methods for assaying the level of candidate gene expression. A variety of methods known in the art may be used to detect assay levels of one or more candidate nucleic acid sequences in a sample. For example, RNA from a cell type or tissue that is known or suspected to express one or more candidate genes of interest may be isolated and tested utilizing hybridization or PCR techniques known in the art. The isolated cells may be, for example, cells derived from a cell culture or from an individual. The analysis of cells taken from a cell culture may be useful, *e.g.*, to test the effect of compounds on the expression of one or more candidate genes, or alternatively, to verify that the cells are ones of a particular cell type that express one or more candidate genes of interest.

As an example, and not by way of limitation, diagnostic methods for the detection of candidate nucleic acids can involve contacting and incubating nucleic acids (including recombinant DNA molecules, cloned genes or degenerate variants thereof) obtained from a sample with one or more labeled nucleic acid reagents, such as recombinant candidate DNA molecules, cloned genes or degenerate variants thereof, under conditions favorable for specifically annealing or hybridizing these reagents to their complementary sequences in the sample nucleic acids. After incubation, all non-annealed or non-hybridized nucleic acids are removed. The presence of nucleic acids that have hybridized, if any such molecules exist, is then detected and the level of candidate nucleic acid sequences to which the nucleic acid reagents have annealed may be compared to the annealing pattern or level expected from a control sample (*e.g.*, from a sample of normal, non-OA cells or tissues) to determine whether candidate nucleic acid is expressed at an elevated level.

In a preferred embodiment of such a detection scheme, the nucleic acid from the cell type or tissue of interest may be immobilized, for example, to a solid support such as a membrane or a plastic surface (for example, on a nylon membrane, a microtiter plate or on polystyrene beads). After incubation, non-annealed, labeled candidate nucleic acid reagents may be easily removed and detection of the remaining, annealed, labeled candidate nucleic acid reagents may be accomplished using standard techniques that are well-known in the art.

Alternative diagnostic methods for the detection of candidate nucleic acids in patient samples or in other cell or tissue sources may involve their amplification, *e.g.*, by PCR (see, for example, the experimental embodiment taught in U.S. Patent No. 4,683,202) followed by detection of the amplified molecules using techniques that are well known to those of skilled in the art. The resulting level of amplified candidate nucleic acids may be compared to those levels that would be expected if the sample being amplified contained only normal levels of the candidate nucleic acid(s), as normal cells or tissues, to determine whether elevated levels of any candidate nucleic acid(s) are expressed.

In one preferred embodiment of such a detection scheme, a cDNA molecule is synthesized from an RNA molecule of interest (*e.g.*, by reverse transcription). A sequence within the cDNA may then be used as a template for a nucleic acid amplification reaction such as PCR. Nucleic acid reagents used as synthesis initiation reagents (*e.g.*, primers) in the reverse transcription and amplification steps of such an assay are preferably chosen from the candidate nucleic acid sequences described herein or are fragments thereof. Preferably, the nucleic acid reagents are at least about 9 to 30 nucleotides in length. The amplification may be performed using, *e.g.*, radioactively labeled or fluorescently labeled nucleotides, for detection. Alternatively, enough amplified product may be made such that the product can be visualized by standard ethidium bromide or other staining methods.

Candidate gene expression assays of the invention may also be performed *in situ* (*i.e.*, directly upon tissue sections of patient tissue, which may be fixed and/or frozen), thereby eliminating the need for nucleic acid purification. Candidate nucleic acid reagents may be used as probes or as primers for such *in situ* procedures (see, for example, Nuovo, PCR In Situ Hybridization: Protocols And Application, 1992, Raven Press, New York). Alternatively, if a sufficient quantity of the appropriate cells can be obtained, standard Northern analysis can be performed to determine the level of candidate gene expression by detecting levels of one or more candidate mRNAs.

Detection of candidate gene products. The diagnostic and prognostic methods of the invention also include ones that comprise detecting levels of a candidate polypeptide and including functionally conserved variants and fragments thereof. For example, antibodies directed against unimpaired, wild-type or mutant candidate gene products or against functionally conserved variants or peptide fragments of a candidate gene product may be used as diagnostic and prognostic reagents. Such reagents may be used, for example, to detect abnormalities in the level of candidate gene product synthesis or expression, or to detect abnormalities in the structure, temporal expression or physical location of a candidate gene product. Antibodies and immunoassay methods such as those described hereinbelow also have important *in vitro* applications for assessing the efficacy of treatments, *e.g.*, for OA. For example, antibodies, or fragments of antibodies, can be used in screens of potentially

therapeutic compounds *in vitro* to ascertain a compound's effects on candidate gene expression and candidate polypeptide production. Compounds that may have beneficial effects on a disorder associated with abnormal candidate gene expression can be identified and a therapeutically effective dose for such compounds may be determined using such assays.

As one example, antibodies or fragments of antibodies may be used to detect the presence of a candidate gene product, a variant of a candidate gene product or fragments thereof, for example, by immunofluorescence techniques employing a fluorescently labeled antibody coupled with light microscopic, flow cytometric or fluorimetric detection methods.

In particularly preferred embodiments, antibodies or fragments thereof may also be employed histologically, for example in immunofluorescence or immunoelectron microscopy techniques, for *in situ* detection of a candidate gene product. *In situ* detection may be accomplished by removing a histological specimen (*e.g.*, a tissue sample) from a patient and applying thereto a labeled antibody of the present invention or a fragment of such an antibody. The antibody or antibody fragment is preferably applied by overlaying the labeled antibody or antibody fragment onto a biological sample. Through the use of such a procedure, it is possible to detect, not only the presence of a candidate gene product, but also the gene product's distribution in the examined tissue. A wide variety of histological methods that are well known in the art (for example, staining procedures) can be readily modified by those skilled in the art without undue experimentation to achieve such *in situ* detection.

Immunoassays for candidate gene products will typically comprise incubating a biological sample (for example, a tissue extract) in the presence of a detectably labeled antibody that is capable of specifically binding a candidate gene product (including, for example, a functionally conserved variant or a peptide fragment thereof). The bound antibody may then be detected by any of a number of techniques well known in the art.

Therapeutic Methods and Pharmaceutical Compositions:

Candidate nucleic acids and polypeptides, and specific antibodies thereto may also be used in therapeutic methods and compositions, *e.g.*, to treat, prevent or ameliorate diseases and disorders associated with abnormal (preferably elevated) levels of the candidate gene's expression. In preferred embodiments such methods are used to treat OA. In one preferred embodiment the therapeutic methods of the invention comprise administering one or more compounds that modulate (*e.g.*, inhibit) the expression or activity of a candidate gene or its gene product; for example, compounds that bind to a candidate nucleic acid or polypeptide of the invention, compounds that modulate expression of a candidate gene, and/or compounds that interfere with or modulate binding of a candidate nucleic acid or polypeptide with a binding compound.

In another preferred embodiment, the therapeutic methods of the invention may comprise one or more cell-targeted therapies which target compounds (for example, drugs, pro-drugs, toxins or cytotoxins) to cells expressing a candidate nucleic acid or polypeptide.

Inhibitory approaches. In alternative embodiments, the present invention provides methods and compositions for treating a disease or disorder (for example, OA) associated with the abnormal expression or activity of a candidate gene or gene product by modulating (*e.g.*, increasing or decreasing) the expression or activity of the candidate gene or its gene product. Such methods may simply comprise administering one or more compounds that modulate expression of a candidate gene, synthesis of a candidate gene product or activity of a candidate gene product so the immune response is modulated (*e.g.*, enhanced or suppressed). Preferably, these one or more compounds are administered until one or more symptoms of the disorder are eliminated or at least ameliorated.

Among the compounds that may exhibit an ability to modulate the activity, expression or synthesis of a candidate nucleic acid are antisense molecules. Such molecules may be designed to reduce or inhibit wild-type nucleic acids and polypeptides or, alternatively, may target mutant candidate nucleic acids or polypeptides.

Antisense RNA and DNA molecules act to directly block the translation of mRNA by hybridizing to target mRNA molecules and preventing protein translation. Antisense approaches involve the design of oligonucleotides that are complementary to a target gene mRNA. The antisense oligonucleotides will bind to the complementary target gene mRNA transcripts and prevent translation. Absolute complementarity, although preferred, is not required. As used in this description, "antisense" broadly includes RNA-RNA interactions, triple helix interactions, ribozymes and RNase-H mediated arrest. Antisense nucleic acid molecules can be encoded by a recombinant gene for expression in a cell (see, *e.g.*, U.S. Patent Nos. 5,814,500; and 5,811,234) or, alternatively, they can be prepared synthetically (U.S. Patent No. 5,780,607).

A sequence that is "complementary" to a portion of a nucleic acid refers to a sequence having sufficient complementarity to be able to hybridize with the nucleic acid and form a stable duplex. The ability of nucleic acids to hybridize will depend both on the degree of sequence complementarity and the length of the antisense nucleic acid. Generally, however, the longer the hybridizing nucleic acid, the more base mismatches it may contain and still form a stable duplex (or triplex in triple helix methods). A tolerable degree of mismatch can be readily ascertained, *e.g.*, by using standard procedures to determine the melting temperature of a hybridized complex.

In one preferred embodiment, oligonucleotides complementary to non-coding regions of a candidate gene may be used in an antisense approach to inhibit translation of endogenous candidate mRNA molecules. Antisense nucleic acids are preferably at least six nucleotides in length, and more preferably range from between about six to about 50 nucleotides in length. In specific embodiments, the oligonucleotides may be at least 10, at least 15, at least 20, at least 25 or at least 50 nucleotides in length.

It is generally preferred that *in vitro* studies are first performed to quantitate the ability of an antisense oligonucleotide to inhibit gene expression. It is preferred that these studies utilize controls that distinguish between antisense gene inhibition and nonspecific biological effects of oligonucleotides. It is also preferred that these studies compare levels of

the target RNA or protein with that of an internal control RNA or protein. Additionally, it is envisioned that results obtained using the antisense oligonucleotide are compared with those obtained using a control oligonucleotide. It is preferred that the control oligonucleotide is of approximately the same length as the test oligonucleotide and that the nucleotide sequence of the oligonucleotide differs from the antisense sequence no more than is necessary to prevent specific hybridization to the target sequence.

While antisense nucleotides complementary to the target gene coding region sequence could be used, those complementary to the transcribed, untranslated region are most preferred.

Antisense molecules are preferably delivered to cells, such as chondrocytes, that express the target gene *in vivo*. A number of methods have been developed for delivering antisense DNA or RNA to cells. For example, antisense molecules can be injected directly into the tissue site (*e.g.*, directly into a tumor), or modified antisense molecules can be designed to target the desired cells (*e.g.*, antisense linked to peptides or antibodies that specifically bind receptors or antigens expressed on the target cell surface) can be administered systemically.

Preferred embodiments achieve intracellular concentrations of antisense nucleic acid molecules which are sufficient to suppress translation of endogenous mRNAs. For example, one preferred approach uses a recombinant DNA construct in which the antisense oligonucleotide is placed under the control of a strong pol III or pol II promoter. The use of such a construct to transfect target cells in the patient will result in the transcription of sufficient amounts of single stranded RNAs that will form complementary base pairs with the endogenous target gene transcripts and thereby prevent translation of the target gene mRNA. For example, a vector, as set forth above, can be introduced *e.g.*, such that it is taken up by a cell and directs the transcription of an antisense RNA. Such a vector can remain episomal or become chromosomally integrated, as long as it can be transcribed to produce the desired antisense RNA. Such vectors can be constructed by recombinant DNA technology methods standard in the art. Vectors can be plasmid, viral, or others known in the

art, used for replication and expression in mammalian cells. Expression of the sequence encoding the antisense RNA can be by any promoter known in the art to act in the particular cell type (for example in a hemopoietic cell). For example, any of the promoters discussed *supra* in connection with the expression of recombinant candidate nucleic acids can also be used to express a candidate antisense nucleic acid.

In addition to antisense technology, RNA aptamers (Good et al., 1997, Gene Therapy 4: 45-54), double stranded RNA (WO 99/32619), ribozymes (Cech. J., 1988, Amer. Med Assn. 260:3030; Cotten et al., 1989, EMBO J. 8:3861-3866; Grassi and Marini, 1996, Annals of Medicine 28: 499-510; Gibson, 1996, Cancer and Metastasis Reviews 15: 287-299) and/or triple helix DNA (Gee, J.E. et al. (1994) In: Huber, B.E. and B. I. Carr, *Molecular and Immunologic Approaches*, Futura Publishing Co., Mt. Kisco, N.Y.) may be used to modulate the activity, expression or synthesis of a target candidate nucleic acid according to methods familiar to one of skill in the art.

Alternatively, small interfering RNA (siRNA) molecules can also be used to inhibit the expression of nucleic acids for a candidate receptor or for a candidate ligand. RNA interference is a method in which exogenous, short RNA duplexes are administered where one strand corresponds to the coding region of the target mRNA (Elbashir *et al.*, *Nature* 2001, 411: 494-498). Upon entry into cells, siRNA molecules cause not only degradation of the exogenous RNA duplexes, but also of single-stranded RNAs having identical sequences, including endogenous messenger RNAs. Accordingly, siRNA may be more potent and effective than traditional antisense RNA methodologies since the technique is believed to act through a catalytic mechanism.

Preferred siRNA molecules are typically greater than about 19 nucleotides in length and comprise the sequence of a nucleic acid for a candidate receptor or its ligand. Effective strategies for delivering siRNA to target cells include any of the methods described, *supra*, for delivering antisense nucleic acids. For example, siRNA can be introduced to cells by transduction using physical or chemical transfection. Alternatively siRNAs may be expressed in cells using, *e.g.*, various PolIII promoter expression cassettes that allow

transcription of functional siRNA or precursors thereof. See, for example, Scherr *et al., Curr. Med. Chem.* 2003, 10(3):245-256; Turki *et al., Hum. Gene Ther.* 2002, 13(18):2197-2201; Cornell *et al., Nat. Struct. Biol.* 2003, 10(2):91-92.

Pharmaceutical preparations. Compositions used in the therapeutic methods of this invention may be administered (*e.g., in vitro* or *ex vivo* to cell cultures, or, more preferably, *in vivo* to an individual) at therapeutically effective doses to treat a disease or disorder such as OA that is associated with abnormal candidate gene expression and/or activity. For example, compounds, including compounds identified in such screening methods as described above, that bind to a candidate gene or gene product of the invention may be administered to the cells or individual so that expression and/or activity of the candidate gene or gene product is inhibited. The invention therefore also provides pharmaceutical preparations for use, e.g., as therapeutic compounds to treat disorders, including OA, that are associated with abnormal candidate gene expression or activity.

The terms "therapeutically effective dose" and "effective amount" refer to the amount of the compound that is sufficient to result in a therapeutic response. In embodiments where a compound (*e.g.*, a drug or toxin) is administered in a complex (*e.g.*, with a specific antibody), the terms "therapeutically effective dose" and "effective amount" may refer to the amount of the complex that is sufficient to result in a therapeutic response. A therapeutic response may be any response that a user (*e.g.*, a clinician) will recognize as an effective response to the therapy. Thus, a therapeutic response will generally be an amelioration of one or more symptoms of a disease or disorder. In preferred embodiments, where the pharmaceutical preparations are used to treat OA, a therapeutic response may be a reduction in the amount of cartilage degradation observed, *e.g.*, in biopsies from a patient during treatment.

Toxicity and therapeutic efficacy of compounds can be determined by standard pharmaceutical procedures, for example in cell culture assays or using experimental animals to determine the LD_{50} and the ED_{50}. The parameters LD_{50} and ED_{50} are well known in the art, and refer to the doses of a compound that are lethal to 50% of a population and

therapeutically effective in 50% of a population, respectively. The dose ratio between toxic and therapeutic effects is referred to as the therapeutic index and may be expressed as the ratio: LD_{50}/ED_{50}. Compounds that exhibit large therapeutic indices are preferred.

While compounds that exhibit toxic side effects may be used, however, in such instances it is particularly preferable to use delivery systems that specifically target such compounds to the site of affected tissue so as to minimize potential damage to other cells, tissues or organs and to reduce side effects.

Data obtained from cell culture assay or animal studies may be used to formulate a range of dosages for use in humans. The dosage of compounds used in therapeutic methods of the present invention preferably lie within a range of circulating concentrations that includes the ED_{50} concentration but with little or no toxicity (*e.g.*, below the LD_{50} concentration). The particular dosage used in any application may vary within this range, depending upon factors such as the particular dosage form employed, the route of administration utilized, the conditions of the individual (*e.g.*, patient), and so forth.

A therapeutically effective dose may be initially estimated from cell culture assays and formulated in animal models to achieve a circulating concentration range that includes the IC_{50}. The IC_{50} concentration of a compound is the concentration that achieves a half-maximal inhibition of symptoms (*e.g.*, as determined from the cell culture assays). Appropriate dosages for use in a particular individual, for example in human patients, may then be more accurately determined using such information.

Measures of compounds in plasma may be routinely measured in an individual such as a patient by techniques such as high performance liquid chromatography (HPLC) or gas chromatography.

Pharmaceutical compositions for use in accordance with the present invention may be formulated in conventional manner using one or more physiologically acceptable carriers or excipients.

Thus, the compounds and their physiologically acceptable salts and solvates may be formulated for administration by inhalation or insufflation (either through the mouth or the nose) or oral, buccal, parenteral or rectal administration.

For oral administration, the pharmaceutical compositions may take the form of, for example, tablets or capsules prepared by conventional means with pharmaceutically acceptable excipients such as binding agents (*e.g.*, pregelatinised maize starch, polyvinylpyrrolidone or hydroxypropyl methylcellulose); fillers (*e.g.*, lactose, microcrystalline cellulose or calcium hydrogen phosphate); lubricants (*e.g.*, magnesium stearate, talc or silica); disintegrants (*e.g.*, potato starch or sodium starch glycolate); or wetting agents (*e.g.*, sodium lauryl sulphate). The tablets may be coated by methods well known in the art. Liquid preparations for oral administration may take the form of, for example, solutions, syrups or suspensions, or they may be presented as a dry product for constitution with water or other suitable vehicle before use. Such liquid preparations may be prepared by conventional means with pharmaceutically acceptable additives such as suspending agents (*e.g.*, sorbitol syrup, cellulose derivatives or hydrogenated edible fats); emulsifying agents (*e.g.*, lecithin or acacia); non-aqueous vehicles (*e.g.*, almond oil, oily esters, ethyl alcohol or fractionated vegetable oils); and preservatives (*e.g.*, methyl or propyl-p-hydroxybenzoates or sorbic acid). The preparations may also contain buffer salts, flavoring, coloring and sweetening agents as appropriate.

Preparations for oral administration may be suitably formulated to give controlled release of the active compound. For buccal administration the compositions may take the form of tablets or lozenges formulated in conventional manner. For administration by inhalation, the compounds for use according to the present invention are conveniently delivered in the form of an aerosol spray presentation from pressurized packs or a nebuliser, with the use of a suitable propellant, *e.g.*, dichlorodifluoromethane, trichlorofluoromethane, dichlorotetrafluoroethane, carbon dioxide or other suitable gas. In the case of a pressurized aerosol the dosage unit may be determined by providing a valve to deliver a metered amount. Capsules and cartridges of *e.g.*, gelatin for use in an inhaler or insufflator may be formulated

containing a powder mix of the compound and a suitable powder base such as lactose or starch.

The compounds may be formulated for parenteral administration by injection, *e.g.*, by bolus injection or continuous infusion. Formulations for injection may be presented in unit dosage form, *e.g.*, in ampoules or in multi-dose containers, with an added preservative. The compositions may take such forms as suspensions, solutions or emulsions in oily or aqueous vehicles, and may contain formulatory agents such as suspending, stabilizing and/or dispersing agents. Alternatively, the active ingredient may be in powder form for constitution with a suitable vehicle, *e.g.*, sterile pyrogen-free water, before use.

The compounds may also be formulated in rectal compositions such as suppositories or retention enemas, *e.g.*, containing conventional suppository bases such as cocoa butter or other glycerides.

In addition to the formulations described previously, the compounds may also be formulated as a depot preparation. Such long acting formulations may be administered by implantation (for example subcutaneously or intramuscularly) or by intramuscular injection. Thus, for example, the compounds may be formulated with suitable polymeric or hydrophobic materials (for example as an emulsion in an acceptable oil) or ion exchange resins, or as sparingly soluble derivatives, for example, as a sparingly soluble salt.

The compositions may, if desired, be presented in a pack or dispenser device that may contain one or more unit dosage forms containing the active ingredient. The pack may for example comprise metal or plastic foil, such as a blister pack. The pack or dispenser device may be accompanied by instructions for administration.

Numerous references, including patents, patent applications and various publications, are cited and discussed in the description of this invention. The citation and/or discussion of such references is provided merely to clarify the description of the present invention and is not an admission that any such reference is "prior art" to the invention

described herein. All references cited and discussed in this specification (including references to biological sequences deposited in GenBank or other public databases) are incorporated herein by reference in their entirety and to the same extent as if each reference was individually incorporated by reference.

EXAMPLES

The present invention is also described by means of the following examples. However, the use of these or other examples anywhere in the specification is illustrative only and in no way limits the scope and meaning of the invention or of any exemplified term. Likewise, the invention is not limited to any particular preferred embodiments described herein. Indeed, many modifications and variations of the invention may be apparent to those skilled in the art upon reading this specification and can be made without departing from its spirit and scope. The invention is therefore to be limited only by the terms of the appended claims along with the full scope of equivalents to which the claims are entitled.

EXAMPLE 1:
A High Throughput Screen to identify candidate genes related to OA employing RT:PCR analysis of OA "marker" genes

This example describes experiments that use a real time polymerase chain reaction (RT-PCR) assay to identify candidate genes or gene products that may be related to the pathogenesis of OA. In particular, the experiments described in this example test individual full length cDNAs in a high throughput parallel mode for their ability to activate one or more marker genes the expression of which is associated with OA in human articular chondrocyte (HAC) cells.

Materials and Methods:

Data mining OA cDNA libraries. cDNA libraries are preferably generated "in house" from OA chondrocyte cells and used in screening assays of the present invention. Raw sequences of genes in the OA cDNA library are pre-processed and then annotated to

identify clones that are likely to be particularly useful as drug targets. In particular, the Phred/Phrap system (Gordon *et al.*, *Genome Re.* 2001, 11(4):614-625; Ewing *et al.*, *Genome Res.* 1998, 8:175-185; Ewing *et al.*, *Genome Res.* 1998, 8:186-194; Gordon *et al.*, *Genome Res.* 1998, 8;195-202) is used to trim raw sequences to high quality regions and to trim vector sequences. Mitochondrial DNA, ribosomal DNA, repeat regions, low complexity sequence and linker regions are removed. Then, the resulting processed sequences are compared to known and predicted genes in the GenBank database.

Next, the resulting sequence annotations are searched for keywords of interest to select specific clones for screening. The keywords are chosen to emphasize proteins in classes considered most likely to play a role in the disease process based on current biological knowledge. Thus, for example, terms indicative of signal transduction and proteolysis (*e.g.*, "kinase," "receptor," "factor" and "protease") are included since these processes have been previously implicated in osteoarthritis. Individual full length clones for genes selected in this way are then retrieved.

Preparation of plasmid DNA from full length cDNA clones. Bacterial stocks of full-length clones from the OA cDNA libraries in pCMVSport6 vector (Invitrogen, Carlsbad CA) are grown in 96 deep-well blocks (Qiagen, Valencia CA), each well containing 1.0 mL of Terrific broth (Sigma, St. Louis MO) and ampicillin (40 μg/mL). The cultures are initially grown for 24 hours at 37 °C with shaking at 300 RPM, re-innoculated into a fresh block and further grown overnight to ensure uniform growth of bacteria in all wells. Plasmid DNA is isolated from the bacteria with a Biorobot 8000 (Qiagen, Valencia CA) following standard protocols described by the manufacturer.

GATEWAY™ transfer of full-length cDNA clones. In order to screen individual clones in an RT-PCR assay, cDNA clones in the OA libraries are transferred from the pCMVSport6 vector to a retroviral vector using the GATEWAY™ platform (Invitrogen, Carlsbad CA).

Gateway BP reactions are carried out in 96-well plates (Ashford, United Kingdom). Briefly, 1.0 μL (100-120 ng) plasmid DNA is added to each well containing 1 μL

(100-120 ng) pDONR 201 entry vector (Invitrogen, Carlsbad CA), 1 μL BP reaction buffer (Invitrogen, Carlsbad CA), 1 μL tris-EDTA and 1 μL BP Clonase enzyme mix (Invitrogen, Carlsbad CA) on ice. The plates are incubated at 25 °C for three hours.

The Gateway LR reaction mix, consisting of 0.25 μL of 0.75 M NaCl, 1.0 μL (100-120 ng) linearized retroviral vector and 1.5 μL LR Clonase enzyme mix (Invitrogen, Carlsbad CA) is added to each BP reaction.

The retroviral vector contains a hybrid cytomegalovirus (CMV)/Maloney murine leukemia virus (MoMuLV) 5' LTR, a MoMuLV 3' LTR and a retroviral packaging Ψ site and may be constructed according to conventional methods. The same vector is also commercially available (Clontech). Samples are mixed thoroughly and incubated for two additional hours at 25 °C. One-tenth volume (0.8 μL; 2 mg/mL) of Proteinase K solution (Invitrogen, Carlsbad CA) is added and incubated at 37 °C for ten minutes.

40 μL of Max efficiency DH5α cells (Invitrogen, Carlsbad CA) are aliquoted into wells of a flat bottom 96-well block (Qiagen, Valencia CA) on ice. 1 μL of the LR reaction mixture from each well is then added to the cells and incubated on ice for 30 minutes. Cells are heat shocked for 30 seconds at 42 °C, placed on ice for 1-2 minutes, and 65 μL of S.O.C. medium (Invitrogen, Carlsbad CA) is added to each well. The 96-well block is incubated at 37 °C for one hour with shaking. 35 μL of the final transformation mixture was added to each well of a 2x48 deep-well block containing LB agar with 40 μg/mL zeocin (Invitrogen, Carlsbad CA), and was grown overnight at 37 °C. Single colonies are inoculated to 1 mL Terrific broth/zeocin (40 μg/mL) in 96-well format and grown overnight at 37 °C/300 RPM. Plasmid DNA is isolated using a Biorobot 8000 (Qiagen, Valencia CA) following standard protocols described by the manufacturer.

Production of Supernatants. GP2-293 packaging cells (BD Biosciences Clontech, Palo Alto CA) are seeded (5 x 10^4 cells per well) in 96-well PDL plates (BD Biosciences Clontech, Palo Alto CA) 16-24 hours prior to transfection in antibiotic-free DMEM containing 10% FBS (Invitrogen, Carlsbad CA). GATEWAY™ constructs along

with envelope vector pVPack-VSV-G (Stratagene, La Jolla CA) are cotransfected into the packaging cells by combining 150 ng GATEWAY™ construct with 150 ng envelope plasmid in a total volume of 25 μL OPTIMEM (Invitrogen, Calsbad CA) in a 96-well format. In a separate plate, 25 μL of OPTIMEM™ is combined with 1 μL of Lipofectamine 2000 reagent (Invitrogen, Carlsbad CA). This second solution is incubated for five minutes at room temperature, and the two solutions are then combined. The DNA-lipofectamine complex is allowed to form for 20 minutes before being added to the cells. The media is replaced with complete media containing antibiotics 16-24 hours after the transfection procedure. The media, containing viral supernatants, is collected at 24 and 48 hours post transfection.

Transduction into Primary Chondrocytes. Primary chondrocytes (isolated from cartilage tissue obtained from joint replacement surgery, Mullenberg Hospital, Plainfield, NJ) are seeded at 1.1 x 10^4 cells per well in duplicate 96-well plates, twenty-four hours prior to transduction. At time of transduction, media are replaced with 100 μL viral supernatant and 100 μL complete media supplemented with 20 mM HEPES and 16 μg/mL polybrene. Cells are centrifuged in a swinging bucket rotor at 32 °C, 1000 x g, for 1.5 hours. The media are replaced after 16-24 hours with fresh media, and cells are incubated for an additional 48 hours.

RNA isolation and RT-PCR. Total cellular RNA is isolated from pooled duplicate 96-well plates using a BioRobot 8000 (Qiagen, Valencia CA) and Qiagen RNeasy 96 Biorobot reagents according to the manufacturer's instructions. On-column DNase I digestion is employed, pursuant to standard protocols published by Qiagen (Valencia CA) to eliminate contaminating genomic DNA. First strand cDNA is synthesized using random primers with a High-Capacity cDNA Archive kit (PE Applied Biosystems, Foster City CA) in a 100 μL reaction volume. Real time PCR (RT-PCR) was performed in a 384-well format on the ABI Prism 7900HT Sequence Detection System (Applied Biosystems, Foster City CA). The cDNA template and PCR mix are distributed using a Biomek FX liquid handling robot. The 20 μL reaction contains 5 μL cDNA, 200 nM forward and reverse primers, and SYBR Green PCR Master Mix (Applied Biosystems, Foster City CA). The default cycling program (95 °C – 10 minutes and 40 cycles of 95 °C – 15 second, 60 °C – 1 minute) is followed by a

dissociation stage whereby a melting curve is generated to confirm the specificity of the PCR product and the absence of primer dimers.

Amplification of the ubiquitously expressed gene GAPDH is used to normalize the amount of cDNA added to the reaction. ROX dye is used as a passive reference to normalize non-PCR related fluctuations in the fluorescence signal. Changes in gene expression are calculated according to the manufacturer's instructions using the comparative C_t method which makes use of a calibrator sample (*i.e.*, a sample to which all others are compared). The value of the calibrator sample is normalized as 1.0 so that expression levels for all other samples are defined as multiples of the expression level measured for the calibrator sample. For RT-PCR experiments described in this example, a retroviral vector containing no cDNA insert is used as the calibrator sample. Briefly, the amount of target relative to the calibrator is calculated according to the formula: $2^{-\Delta\Delta C_T}$ where Ct =thresh hold cycle (cycle# at which the amount of amplified target reaches a fixed thresh hold).

Cell treatment. To optimize RT-PCR conditions and validate the markers chosen in these screens, human articular chondrocytes from knee joint cartilage obtained in joint replacement surgeries are plated in 96 well plates (11,000 cells per well) using DMEM medium containing 10% FBS (Invitrogen, Carlsbad CA). Two days later, the cells are treated with IL-1 (5 ng/mL) (Peprotech,UK, London) and OSM (50 ng/mL) or PDGF (50 ng/mL) or TGF-β (50 ng/mL) overnight in serum free medium. . OSM, PDGF and TGF-β are purchased from R&D systems, (Minneapolis, MN). RNA is isolated from these cells and evaluated by RT-PCR using the methods described above.

Data mining for OA associated genes. Early and late OA cDNA libraries are mined to identify the most abundant genes associated with OA cartilage. Among the most highly expressed genes in early OA libraries is C17. An exemplary nucleotide sequence for this gene is available from GenBank Accession No. NM_018659. The C17 gene encodes a protein that has been described as "cytokine-like" and was previously believed to be expressed only in CD34+ hematopoietic cells. The number of ESTs for C17 is higher in

early OA than in late OA, suggesting that the expression level of this gene decreases during progression of the disease.

A second abundant gene, known as SMOC2 (available from GenBank Accession No. NM_022138) is highly expressed in late OA cartilage, as evidenced by the higher number of ESTs in a late OA cDNA library than in an early OA cDNA library. Thus, expression of this gene presumably increases during progression of the disease.

OA associated genes are also identified by mining gene expression data generated using DNA microarrays. U95A GeneChips from Affymetrix (Santa Clara, CA) are used according to the manufacturer's recommended protocol to compare sets of expressed genes in knee cartilage from 12 OA and 9 healthy patients. The average difference in intensity is calculated for all genes, and the significance of the difference between diseased and healthy patients is evaluated using a statistical t-test. Visual inspection confirms that the computed differences represent differences between patient groups rather than variability in the data. Among the most significantly changed genes between normal and OA knees are the genes OSF-2 (also known as periostin), MARCKS (myristoylated alanine-rich protein kinase C substrate), retinoic acid receptor beta, zinc finger protein Zic1, BASP1 (brain abundant membrane attached signal protein 1), and DIM1.. All of these genes are upregulated in OA patients but have not previously been associated with that disease. GenBank Accession numbers for preferred nucleotide sequences sequences of these genes are provided below, along with GenBank Accession numbers for amino acid sequences that are encoded by these nucleic acids.

TABLE I NEWLY IDENTIFIED OA MARKER GENES

	GenBank Accession Nos.		
Gene	**Nucleotide**	**Protein**	
OSF-2	NM_006475 SEQ ID NO 171	NP_006466	SEQ ID NO 23
MARCKS	NM_002356	NP_002347	SEQ ID NO 24

	SEQ ID NO 172		
Retinoic Acid Receptor β	NM_00965	NP_000956	SEQ ID NO 25
	SEQ ID NO 173;	NP_057236	SEQ ID NO 26
	NM_016152		
	SEQ ID NO 174		
BASP1	NM_006317	NP_006308	SEQ ID NO 27
	SEQ ID NO 175		
Zic1	NM_003412	NP_003403	SEQ ID NO 28
	SEQ ID NO 176		
DIM1	NM_006701	NP_006692	SEQ ID NO 29
	SEQ ID NO 177		

Choosing OA markers. To identify genes that are involved in osteoarthritis (OA) and/or may be useful for the diagnosis or treatment of that disease, a real time polymerase chain reaction (RT-PCR) based assay is used to screen cDNA clones in a high throughput parallel mode. In particular, the assays described in this example use RT-PCR to measure expression of certain genes that are considered "markers" or indicators of OA.

The marker geness are preferably selected to represent various biological pathways that are affected in OA (see Table II). The GenBank Accession Number for an exemplary nucleotide sequence is also provided for each marker gene. In addition, the gene GAPDH (GenBank Accession No.. AJ_005371) is selected as a ubiquitously expressed "housekeeping" gene to which all samples are normalized.

TABLE II: MARKER GENES FOR OA PHENOTYPES

OA Pheotype/Characteristic	Marker Gene	Accession No.
• Cartilage degradation	Aggrecanase-1	AF148213
	MMP-13	XM_006274
• Aberrant chondrocyte cell differentiation (hypertrophy and proliferation)	Collagen Type I	AF017178
	Collagen Type Iia	XM_012271
	Collagen Type X	NM_000493
• Inflammation	Inos	AB022318
	Cox-2	M90100
• Matrix synthesis	Aggrecan	X80278
	Decorin	AF91944

PCR primers for each of the marker genes is designed with Primer Express software (Applied Biosystems, Foster City CA) under default parameters and reaction conditions. The primer sequences used for marker genes in this example are provided in Table III, below.

TABLE III: RT-PCR PRIMERS TO DETECT OA MARKER GENES

Marker Gene	Primer	Sequence	
Aggrecanase-1	forward	5'-TTTCCCTGGCAAGGACTATGA-3'	(SEQ ID NO:1)
	reverse	5'-AATGGCGTGAGTCGGGC-3'	(SEQ ID NO:2)
MMP-13	forward	5'-TGATCTCTTTTGGAATTAAGGAGCAT-3'	(SEQ ID NO:3)
	reverse	5'-ATGGGCATCTCCTCCATAATTTG-3	(SEQ ID NO:4)
COX-2	forward	5'-AAATTGCTGGCAGGGTTGC-3'	(SEQ ID NO:5)
	reverse	5'-TTTCTGTACTGCGGGTGGAAC-3'	(SEQ ID NO:6)
iNOS	forward	5'-GCAAACCTTCAAGGCAGCC-3'	(SEQ ID NO:7)
	reverse	5'-TGCTGTTTGCCTCGGACAT-3'	(SEQ ID NO:8)
Collagen IIa	forward	5'-ACGCTGCTCGTCGCCG-3'	(SEQ ID NO:9)
	reverse	5'-GCCAGCCTCCTGGACATCCT-3'	(SEQ ID NO:10)
Collage X	forward	5'-ACCCAACACCAAGACACAGTTCT-3'	(SEQ ID NO:11)
	reverse	5'-TCTTACTGCTATACCTTTACTCTTTATGGTGTA-3'	(SEQ ID NO:12)
Collagen I	forward	5'-CAGCCGCTTCACCTACAGC-3'	(SEQ ID NO:13)
	reverse	5'-TTTTGTATTCAATCACTGTCTTGCC-3'	(SEQ ID NO:14)
Decorin	forward	5'-GCCAGCCTCCTGGACATCCT-3'	(SEQ ID NO:15)
	reverse	5'-AGTCCTTTCAGGCTAGCTGCATC-3'	(SEQ ID NO:16)
Aggrecan	forward	5'-TCGAGGACAGCGAGGCC-3'	(SEQ ID NO:17)

	reverse	5'-TCGAGGGTGTAGCGTGTAGAGA-3'	(SEQ ID NO:18)
GAPDH	forward	5'-ATGGGGAAGGTGAAGGTCG-3'	(SEQ ID NO:19)
	reverse	5'-TAAAAGCAGCCCTGGTGACC-3'	(SEQ ID NO:20)

Expression changes of OA markers. To validate the RT-PCR conditions and primers, human articular chondrocyte cells are treated with various compounds as described in the Materials and Methods section, above, for this example. These compounds are known to induce an OA phenotype in the chondrocyte cells. See, for example, Smith *et al.*, *Arthritis Rheum.* 1991, 34:697-706; Tardif *et al.*, *Arthritis Rheum.* 1999, 42:1147-1158.

RT-PCR is performed to determine whether there is any detectable change in expression of one or more marker genes. Table IV, below, summarizes exemplary changes in mRNA levels of each marker mediated by treatment of the chondrocyte cells with: (i) IL-1 and OSM; (ii) TGF-β; and (iii) PDGF. Expression levels are indicated as the multiples of normalized expression levels (*i.e.*, as the "fold changes" in Mrna levels) measured in untreated chondrocyte cells. The data in Table IV indicates that the various OA marker genes undergo the expected changes in their expression levels in response to known treatments that induce an OA phenotype. Moreover, the response of these OA marker genes is sensitive enough to validate this RT-PCR assay for running high throughput functional screens.

TABLE IV: CHANGE OF MARKER GENE EXPRESSION IN TREATED CHONDROCYTE CELLS

	Treatment			
Marker Gene	**IL-1/OSM**	**TGF-β**	**PDGF**	**Untreated**
Aggrecanase-1	50.21	3.81	2.46	1.00
MMP-13	125.37	6.92	4.20	1.00
Collagen Iia	-227.54	1.45	-2.04	1.00
Collagen X	-3.71	19,97	-1.79	1.00
Collagen I	-3.58	3.84	-1.89	1.00

To further validate the RT-PCR assay for use in functional screens, the constitutively active gene AKT/PKB (GenBank Accession No. NPL-001907) is overexpressed in chondrocyte cells by retroviral-mediated gene transfer. Activation of this gene's biochemical pathway induces Aggrecanse-1 and MMP-13 in chondrocyte cells. Cellular RNA is harvested 48 hours and 72 hours post transduction, and changes in the expression of MMP-13 and aggrecanse-1 Mrna are detected by RT-PCR. AKT over-expression results in a 12-fold induction of Aggrecanase-1 and a 9-fold induction of MMP-13.

These experiments validate RT-PCR as a valid and sensitive method that can be used in high throughput functional assays to identify novel mediators of an OA phenotype.

Results:

Verified hits from an RT-PCR screen. The high throughput screen disclosed in this example is performed by overexpressing a select set of about 1200 test genes mined from OA libraries in primary chondrocytes. Expression levels of the OA marker genes are measured by RT-PCR when these test genes are expressed in chondrocyte cells, and these expression levels are compared to the expression levels measured in untransformed cells. To the Applicant's knowledge, heretofore, high throughput screens of chondrocytes have not been reported.

Table V lists 63 candidate genes identified in such an RT-PCR screen, along with GenBank accession numbers for their preferred nucleotide sequences. Residues coding the predicted amino acid sequence (*i.e.*, the coding sequence or "CDS") are also specified.

TABLE V: CANDIDATE GENES IDENTIFIED IN RT-PCR SCREEN

Gene	SEQ ID NOS	Accession # (nucleotide)	CDS	Accession # (protein)
SFRS3	30/31	NM_003017	106-600	NP_003008
SFRS10	32/33	NM_004593	122-988	NP_004584
U2AF1	34/35	NM_006758	39-761	NP_006749
TGFBR2	36/37	NM_003242	336-2039	NP_003233
TSC22	38/39	NM_006022	192-626	NP_006013
MTIF3	40/41	NM_152912	237-1073	NP_690876
CAMK2G	42/43	XM_044349	5-1561	XP_044349

Gene	SEQ ID NOS	Accession # (nucleotide)	CDS	Accession # (protein)
PHKG1	44/45	NM_006213	120-1283	NP_006204
DTR	46/47	NM_001945	262-888	NP_001936
TGFA	48/49	NM_003236	32-514	NP_003227
SF3B1	50/51	NM_012433	1-3915	NP_036565
BCAT1	52/53	BC033864	424-1386	AAH33864
CSNK2A1	54/55	NM_001895	149-1324	NP_001886
FLJ14103	56/57	NM_024689	76-624	NP_078965
U5snRNP-AP	58/59	AF221842	106-2931	AAF66128
VTI2	60/61	NM_006370	341-1039	NP_006361
LOC51231	62/63	NM_016440	119-1543	NP_057524
TCEA3	64/65	XM_114075	136-1182	XP_114075
UBE2G1	66/67	NM_003342	167-679	NP_003333
SENP3	68/69	NM_015670	71-1795	NP_056485
SF3A3	70/71	NM_006802	9-1514	NP_006793
NRF1	72/73	NM_005011	79-1647	NM_005002
ARF6	74/75	NM_001663	518-1045	NP_001654
TNFSF12	76/77	NM_003809	97-846	NP_003800
RELA	78/79	NM_021975	39-1652	NP_068810
TNFRSF1A	80/81	NM_001065	282-1649	NP_001056
RPS6KB2	82/83	NM_003952	16-1503	NP_003943
GSK3A	84/85	NM_019884	115-1566	NP_063937
CLC	86/87	NM_013246	46-723	NP_037378
ZNF213	88/89	XM_036493	366-1745	XP_036493
CAMK1	90/91	NM_003656	179-1291	NP_003647
FGFR1	92/93	NM_023107	727-1635	NP_075595
CLK1	94/95	NM_004071	156-1610	NP_004062
MUS81	96/97	NM_025128	511-1941	NP_079404
VEGF	98/99	NM_003376	702-1277	NP_003367
FGF18	100/101	NM_033649	538-1161	NP_387498
HGS	102/103	NM_004712	78-2411	NP_004703
RIPK2	103/104	NM_003821	212-1834	NP_003812
TNFAIP1	105/106	NM_021137	212-1162	NP_066960
CLK3	107/108	NM_003992	57-1529	NP_003983
ADAMTS9	109/110	NM_020249	13-3231	NP_064634
CDKN2C	111/112	NM_001262	1217-1723	NP_001253
FYN	113/114	NM_002037	580-2193	NP_002028
FGF1	115/116	NM_000800	142-609	NP_000791
PTN	117/118	NM_002825	396-902	NP_002816
GLA	119/120	NM_000169	61-1350	NP_000160
LOC162542	121/122	XM_091624	12-287	XP_091624
EXT2	123/124	NM_000401	488-2644	NP_000392
METAP2	125/126	NM_006838	35-1471	NP_006829

Gene	SEQ ID NOS	Accession # (nucleotide)	CDS	Accession # (protein)
MLL3	127/128	NM_021230	364-12441	NP_067053
RARG	129/130	NM_000966	138-1502	NP_000957
Rho GEF p114	131/132	NM_015318	108-3155	NP_056133
CHKL	133/134	NM_005198	185-1372	NP_005189
ANXA2	135/136	NM_004039	50-1069	NP_004030
LOC143785	137/138	XM_084635	390-1025	XP_084635
TGFB3	139/140	NM_003239	254-1492	NP_003230
MAP3K11	141/142	NM_002419	494-3037	NP_002410
PHKG2	143/144	NM_000294	94-1314	NP_000285
NNMT	145/146	NM_006169	118-912	NP_006160
TPT1	147/148	NM_003295	95-613	NP_003286
IL17BR	149/150	NM_018725	45-1553	NP_061195
ECRG4	151/152	NM_032411	109-555	NP_115787

EXAMPLE 2:
A High Throughput Screen to identify candidate genes related to OA employing analysis of clonal proliferation of chondrocyte clusters in vitro

This example describes experiments using another high throughput screen to identify genes and gene products associated with OA. In particular, the experiments described in this example screen whole cDNA libraries and identify genes that induce clonal proliferation of chondrocyte clusters, a type of cell proliferation associated with osteoarthritic chondrocytes .

Materials and Methods:

Construction of late-OA cDNA library. 1 μg of polyA(+) RNA is isolated from 200 μg of total RNA (extracted from OA chondrocyte cells) using a Dynabeads mRNA Purification kit (Dynal, Lake Success NY) following the manufacturer's recommend protocol. The library is constructed using the Superscript Choice System for cDNA Synthesis (Invitrogen Life Technologies, Carlsbad CA). The procedure follows the manufacturer's recommended protocol, but with the modifications specifically noted here. A modified oligo d(T)-NotI primer is used to prime the first-strand synthesis reaction.

Following second-strand synthesis, adaptor ligation includes the use of EcoRI half-site adapters and Not I restriction digest to allow for the directional cloning of the size fractionated double-stranded cDNA into the entry vector pENTR2B (Invitrogen Life Technologies, Carlsbad CA). This vector is constructed to contain GATEWAY™ site-specific recombination sites (attL1 and attL2) flanking the cloned cDNAs and allows the one-step transfer of cDNA inserts into retroviral vectors containing the attR1 and attR2 site-specific recombination sites via LR clonase.

Transfer of Late-OA library. 300 ng of amplified library DNA is used for the transfer of cDNAs into each of two linearized retroviral vectors using LR Clonase (Invitrogen, Carslbad CA) according to the manufacturer's recommended protocol. Following a brief clean-up step, the LR reaction products are electroporated into STBL4 electrocompetent cells (Invitrogen Life Technologies, Carlsbad CA) and amplified on selective solid medium.

Construction of Early-OA cDNA library. cDNA libraries are constructed "in house" from chondrocytes isolated from early stage human OA cartilage, following the same procedure as for the late-OA cDNA library, above, but with the following exceptions. A modified oligo d(T)-Sfil(B) primer primes the first strand synthesis reaction. Following second-strand synthesis, adaptor ligation includes the use of Sfi I (A) half-site adapters and Sfi I restriction digest to allow for the directional cloning of the size fractionated double-stranded cDNA into the vector pCMBSport6 (Invitrogen Life Technologies, Carlsbad CA). This vector has been constructed to contain the GATEWAY™ site-specific recombination sites attB1 and attB2 flanking the cloned cDNAs and requires a two-step transfer of cDNA inserts — first into an entry vector (BP reaction) and second into a retroviral vector containing the attR1 and attR2 site-specific recombination sites via LR cleanse (LR Reaction; Nitrogen, Carlsbad CA). The early-OA cDNA library is transferred into a retroviral vector using DH10B cells from Invitrogen (Carlsbad, CA) as the host *E. coli* strain.

Transfection. GP2-293 cells are plated the day before transfection at 7 x 10^5 cells per well in 6-well Bio coat plates (BD Biosciences, Palo Alto CA) with 2 M DMEM

containing 10% FBS per well (Nitrogen, Carlsbad CA). The following day, for each well to be transfected, 1 μg of OA cDNA library DNA and 1 μg of pVpack-VSVG plasmids are diluted in OPTIMEM™ medium (Invitrogen, Carlsbad CA) to a final volume of 250 μL.

Lipofectamine 2000 (Invitrogen, Carlsbad CA) (9 μL/2 μg DNA for each well) is diluted in OPTIMEM™ to 250 μL final volume. The diluted Lipofectamine is added drop wise to the diluted DNA, gently mixed and incubated at room temperature for 20 minutes. The DNA-Lipofectamine complex (500 μL per well) is then added directly into the 2 mL conditioned medium, and the plates are incubated overnight at 37 °C. The following day, the medium in each well is aspirated and replaced with 3 mL DMEM containing 10% FBS per well. Supernatants are collected 48 hours and 72 hours post transfection, filtered through a 0.22 micron filter and frozen at -80 °C.

Spinfection of viral supernatants into chondrocytes. Human chondrocyte cells (Cell Applications, San Diego CA) derived from fetal human cartilage are cryopreserved at the first passage and used at passage 2. The chondrocyte cells are cultured in six well plates at a cell density of 2.5 x 10^5 cells per well. The complete growth media is replaced with spinnoculation medium containing DMEM, 10% FBS, 8 μg/mL polybrene and 10 μM HEPES). The viral supernatants are diluted 1:2 with this medium, filtered through a 0.22 micron filter and added to the wells (2 mL/well). The chondrocyte cells are centrifuged for 1.5 hours at 2700 rpm, 32 °C. The cells are then placed in a CO_2 incubator for six hours. At the end of the day, 2 mL fresh spinnoculation media is added and the cells are incubated overnight. The next day, the spinnoculation media is replaced with growth media (containing DMEM and 10% FBS), and the cells are cultured for three days.

Chondrocyte cloning assay. Three days post transduction, the chondrocyte cells are trypsinized and suspended in 0.4% low melt agarose (Life Technologies, Rockville MD) in complete DMEM (Invitrogen, Carlsbad CA) at a density of 1 x 10^4 cells/mL. 8 mL of the chondrocyte cell suspension is pipeted into 10 cm tissue culture plates that have been previously coated with 8 mL of 0.7% low melt agarose in DMEM containing 10% FBS

(Invitrogen, Carlsbad CA). The agarose is allowed to solidify at room temperature, and then placed in a 37 °C humidified incubator for 3-4 weeks.

Identification of chondrocyte cell clones. Chondrocyte cell clones are identified using a microscope under 20X magnification, picked using a hand pipetor and the seeded directly into 6-well cluster plates (BD Biosciences Clontech, Palo Alto CA) at one clone per well. Clones are allowed to expand in monolayer culture (DMEM, 10% FBS) until confluent.

RNA is isolated using RNeasy 96 (Qiagen, Valencia CA). RT-PCR is performed using 96 well format Advantage RT-PCR kit (Clontech, Palo Alto CA) with Amplitaq Gold (Perkin Elmer, Palo Alto CA), with the following primers for the AttB sites flanking each cDNA:

AttB1	5'-CAAGTTTGTACAAAAAAGC-3'	(SEQ ID NO:21)
AttB2	5'-ACCACTTTGTACAAGAAAG-3'	(SEQ ID NO:22)

The cDNA sequences thus isolated are cloned using a TOPO TA cloning kit (Invitrogen, Carlsbad CA). The plasmid DNA is then sequenced by standard sequencing methods (Seqwright, Houston TX) for identification. Full length clones corresponding to the identified genes were obtained from a full length clone collection that is generated "in house" by routine methods.

GATEWAY™ transfer of full-length clones. Full length cDNA clones obtained from an in house collection and are transferred into a retroviral vector using the Gateway™ platform as described above, and the fidelity of all clones is verified by nucleotide sequencing (Seqwright, Houston TX).

Image Analysis. Validation of a cDNA's ability to promote clonal chondrocyte cell proliferation is measured in agarose cultures of single-gene transduced chondrocyte cells. Chondrocyte cell response is based on the number of clones formed that are greater than 50 microns in diameter. Chondrocyte cell clones are observed using an

Olympus IX70 inverted microscope with a 4X objective (Olympus America, Inc.; Melville NY) under brightfield illumination. Each culture dish is photographed at five different microscope fields on three replicate plates and digitally captured on an Olympus MagnaFire camera and software. Each image is then analyzed using Image-Pro Plus v.4.5 software (Media Cybernetics, Inc., (Silver Spring, MD). Each image is optimized for cell and clone recognition using enhancement filters before being counted. Counts are done automatically using the software, and mean diameter measurements of cells or clones were compiled on an Excel (Microsoft Corporation, Redmond WA) spreadsheet.

Results:

Normal chondrocyte cells quickly lose their phenotype and become fibroblastic when grown in monolayer cultures. However, when grown in a 3-dimensional matrix (*e.g.*, of agarose or alginate) these cells remain chondrocytic in their appearance, gene expression profile and low cell division rate. See, Benya & Shaffer, *Cell* 1982, 30:215-224; Glowacki *et al.*, *Proc. Soc. Exp. Biol. and Med.* 1983, 172:93-98. Under these culturing conditions, certain growth factors have been shown to induce cell proliferation, as evidenced by the formation of cell clusters reminiscent of the clusters observed in OA cartilage. Kato *et al.*, *J. Cell Physiol.* 1987, 133:491-498; Iwamoto *et al.*, *Biochem. Biophys. Res. Comm.* 1989, 159:1006-1011.

To evaluate whether such growth characteristics could be used in a functional screening assay, the clone forming activity of transduced chondrocyte cells overexpressing bFGF is compared to clone forming activity in chondrocyte cells cultured with bFGF exogenously added to the culture medium. The results demonstrate that expression of a retrovirally transduced gene in chondrocyte cells can stimulate cell proliferation in a manner similar to that observed when the gene product is added exogenously (data not shown).

cDNA libraries are constructed from both early and late stage OA cartilage tissue and transferred to retroviral vectors. These libraries can be virally packed and transduced in early passage human chondrocyte cells. Following growth in suspension cultures for 3-4 weeks, cell clusters are isolated using a micropipet under magnification. The

transgenes are recovered from these cell clusters using PCR, and are identified by routine nucleotide sequencing. The recovered transgenes are preferably verified by determining whether they induce chondrocyte cluster formation when the full length genes are over expressed individually in chondrocyte cells.

Table VI, below, lists candidate genes that can be identified and verified by such a screening assay. GenBank accession numbers for the preferred nucleotide sequences of those genes are also specified, along with the residues coding the predicted amino acid sequence (*i.e.*, the "CDS") accession numbers for preferred amino acid sequences of their gene product(s).

TABLE VI: CANDIDATE GENES IDENTIFIED IN CLONAL SCREENS

Gene	SEQ ID NOs	Accession # (nucleotide)	CDS	Accession # (protein)
C1r	153/154	NM_001733	52-2169	NP_001724
NDUFV2	155/156	NM_021074	19-768	NP_066552
BPOZ	157/158	NM_032548	505-1515	NP_115937
IL17-RC	159/160	NM_032732	198-1814	NP_116121
COMP	161/162	NM_000095	26-2219	NP_000086
SLC16A3	167/168	NM_004207	1-465	NP_004198
FGF1	169/170	NM_000800	142-609	NP_000791

Candidate genes which may be identified in such clonal screening assays include the bFGF gene, further validating the screening assay.

EXAMPLE 3

Sequences for candidate genes and newly identified OA marker genes identified herein

>gi|24025684|gb|NM_003017.2|SFRS3 1403bp mRNA Homo sapiens splicing factor, arginine/serine-rich 3 (SFRS3), mRNA.
CCGGGTGAGTGAGAGAGTTGGTTGGTGTTGGGCCGGAGGAAAGCGGGAAGACTCATCGGA
GCGTGTGGATTTGAGCCGCCGCATTTTTTAACCCTAGATCTCGAAATGCATCGTGATTCC
TGTCCATTGGACTGTAAGGTTTATGTAGGCAATCTTGGAAACAATGGCAACAAGACGGAA
TTGGAACGGGCTTTTGGCTACTATGGACCACTCCGAAGTGTGTGGGTTGCTAGAAACCCA
CCCGGCTTTGCTTTTGTTGAATTTGAAGATCCCCGAGATGCAGCTGATGCAGTCCGAGAG

CTAGATGGAAGAACACTATGTGGCTGCCGTGTAAGAGTGGAACTGTCGAATGGTGAAAAA
AGAAGTAGAAATCGTGGCCCACCTCCCTCTTGGGGTCGTCGCCCTCGAGATGATTATCGT
AGGAGGAGTCCTCCACCTCGTCGCAGATCTCCAAGAAGGAGAAGCTTCTCTCGCAGCCGG
AGCAGGTCCCTTTCTAGAGATAGGAGAAGAGAGAGATCGCTGTCTCGGGAGAGAAATCAC
AAGCCGTCCCGATCCTTCTCTAGGTCTCGTAGTCGATCTAGGTCAAATGAAAGGAAATAG
AAGACAGTTTGCAAGAGAAGTGGTGTACAGGAAATTACTTCATTTGACAGGAGTATGTAC
AGAAAATTCAAGTTTTGTTTGAGACTTCATAAGCTTGGTGCATTTTTAAGATGTTTTAGC
TGTTCAAATCTGTTTGTCTCTTGAAACAGTGACACAAAGGTGTAATTCTCTATGGTTTGA
AATGGATCATACGAGGCATGTAATACCAAGAATTGTTACTTTACAATGTTCCCTTAAGCA
AAATTGAATTTGCTTTGAACTTTTAGTTATGCACAGACTGATAATAAACCTCTAAACCTG
CCCAGCGGAAGTGTGTTTTTTTTTAAATTTAAATACAGAAACAACTGGCAAAAATTGAAC
TAAGATTTACTTTTTTTTCCATAGCTGGGATATAGGCTGCAGCTATAGTTGAACAAGCAG
TCTTTAAAAACTGCTGTGAAACACAGGCCATCAGGGAAAACGAAATGCTGCACTATTAAA
TTAGAGGTTTTTGAAAAATCCAACTCTCATCCTGGGCAGAGGTTGCCTAGTTGGTATAGA
ATGTTAAGTTTCAAGAAAGTTTACCTTTGCTTTAGGTCATAAGTTCCTTATTTGATTGCT
GTATATGGATACATGGCTGTTCGTGACATTCTTTATGTGCAAATTTGTGATTTCAAAAAT
GTCCTGCCAGTTTAAGGGTACATTGTAGAGCCGAACTTTGAGTTACTGTGCAAGATTTTT
TTTTCATGCTGTCATTTGTAATATGTTTTGTGAGAATCCTTGGGATTAAAGTTTTGGTTA
CAAATTGTTAAAAAAAAAAAAAA
>gi|4506901|gb|NP_003008.1|SFRS3 164aa linear splicing factor,
arginine/serine-rich 3; splicing factor, arginine//serine-
rich, 20-kD [Homo sapiens].
MHRDSCPLDCKVYVGNLGNNGNKTELERAFGYYGPLRSVWVARNPPGFAFVEFEDPRDAA
DAVRELDGRTLCGCRVRVELSNGEKRSRNRGPPPSWGRRPRDDYRRRSPPPRRRSPRRRS
FSRSRSRSLSRDRRRERSLSRERNHKPSRSFSRSRSRSRSNERK
>gi|4759097|gb|NM_004593.1|SFRS10 1972bp mRNA Homo sapiens
splicing factor, arginine/serine-rich 10 (transformer 2
homolog, Drosophila) (SFRS10), mRNA.
GAATTCGGCACGAGGGCGACCGGCGCGTCGTGCGGGGCTGCGGCGGAGCCTCCTTAAGGA
AGGTGCAAGAGGTTGGCAGCTTCGATTGAAGCACATCGACCGGCGACAGCAGCCAGGAGT
CATGAGCGACAGCGGCGAGCAGAACTACGGCGAGCGGGAATCCCGTTCTGCTTCCAGAAG
TGGAAGTGCTCACGGATCGGGGAAATCTGCAAGGCATACCCCTGCAAGGTCTCGCTCCAA
GGAAGATTCCAGGCGTTCCAGATCAAAGTCCAGGTCCCGATCTGAATCTAGGTCTAGATC
CAGAAGAAGCTCCCGAAGGCATTATACCCGGTCACGGTCTCGCTCCCGCTCCCATAGACG
ATCACGTAGCAGGTCTTACAGTCGAGATTATCGTAGACGGCACAGCCACAGCCATTCTCC
CATGTCTACTCGCAGGCGTCATGTTGGGAATCGGGCAAATCCTGATCCTAACTGTTGTCT
TGGAGTATTTGGGCTGAGCTTGTACACCACAGAAAGAGATCTAAGAGAAGTGTTCTCTAA
ATATGGTCCCATTGCCGATGTGTCTATTGTATATGACCAGCAGTCTAGGCGTTCAAGAGG
ATTTGCCTTTGTATATTTTGAAAATGTAGATGATGCCAAGGAAGCTAAAGAACGTGCCAA
TGGAATGGAGCTTGATGGGCGTAGGATCAGAGTTGATTTCTCTATAACAAAAAGACCACA
TACGCCAACACCAGGAATTTACATGGGGAGACCTACCTATGGCAGCTCTCGCCGTCGGGA
TTACTATGACAGAGGATATGATCGGGGCTATGATGATCGGGACTACTATAGCAGATCATA
CAGAGGAGGAGGTGGAGGAGGAGGAGGATGGAGAGCTGCCCAAGACAGGGATCAGATTTA
TAGAAGGCGGTCACCTTCTCCTTACTATAGTCGTGGAGGATACAGATCACGTTCCAGATC
TCGATCATACTCACCTCGTCGCTATTAAAGCATGAAGACTTTCTGAAACCTGCCCTAGAG
CTGGGATATTGTTTGTGGGCAATATTTTTTATTGTCTCTTGTTTAAAAAGTGAACAGTGC
CTAGTGAAGTTAGGTGACTTTTACACCTTTTACGATGACTACTTTTGGTGGAGTTGAAAT

GCTGTTTTCATTCTGCATTTGTGTAGTTTGGTGCTTTGTTCCAAGTTAAGTGTTTTCAGA
AAAGTATGTTTTGCATGTATTTTTTTACAGTCTAAATTTTGACTGCTGAGAAGTTTCTAT
TGTACAAAACTTCATTTAAAAGGTTTTTCTACTGAATCCAGGGTATTCTGAAGATCGAAG
CCTGTGTAAAATGCTACCAAATGGCAAAAAGCAACAATAAACAGTTTGATTTTTACTTTT
CTTTCTAACATATCAATGCTTAGCAGAACTATTCAGATTGTCAGTAGTAAATTTAAAGAC
AAATGCCCGTTTTCCTCCAGTCCATGAAACATACCATACTTATATACCTGCAACTAAGTG
TTTAAAATTATGCTCTGTAACTCTGTACTGCTAGTATTAGAACTAAAAATCTTAAAATAC
AGCCAGTGCTTAATGCTTATATCAATGTGGATTTGTCGGCTTTTATGTAATCTGTAATAT
GTATAGCAGGAAATACGAAGAGTTACACAGTGTATGCCTTAAAAGGCTGTTTCTTAAAGG
TGTTACAAGGGGATAATGGTATTTCAACTAGTTATCAGCAAGTGACAATACATTCCACCA
CAAATACACTCTTGTTCTTCTAGCTTTTAGACTATATGAAAAAACCGGGTGCTTCAAAGT
ACATGATAAGGGAACACTATACCTGTCATGGATGAACTGAAGACTTTGCCTGTTCATTTT
TTAAATATTATTTTCAGGTCCTTTGCTTACCAAAGGAGGCCCAATTTCACTCAAATGTTT
TGAGAACTGTGTTTAAATAAACGCAAATGAAAAGAAAAAAAAAAAAAAAAAA
>gi|4759098|gb|NP_004584.1|SFRS10 288aa linear splicing
factor, arginine/serine-rich 10 (transformer 2 homolog,
Drosophila); splicing factor, arginine/serine-rich
(transformer 2 Drosophila homolog) 10 [Homo sapiens].
MSDSGEQNYGERESRSASRSGSAHGSGKSARHTPARSRSKEDSRRSRSKSRSRSESRSRS
RRSSRRHYTRSRSRSRSHRRSRSRSYSRDYRRRHSHSHSPMSTRRRHVGNRANPDPNCCL
GVFGLSLYTTERDLREVFSKYGPIADVSIVYDQQSRRSRGFAFVYFENVDDAKEAKERAN
GMELDGRRIRVDFSITKRPHTPTPGIYMGRPTYGSSRRRDYYDRGYDRGYDDRDYYSRSY
RGGGGGGGGWRAAQDRDQIYRRRSPSPYYSRGGYRSRSRSRSYSPRRY
>gi|5803206|gb|NM_006758.1|U2AF1 904bp mRNA Homo sapiens
U2(RNU2) small nuclear RNA auxillary factor 1 (U2AF1), mRNA.
GGAATTCCGTCGACGGCAGCGGCGGCGGCGGGTGGGAAATGGCGGAGTATCTGGCCTCCA
TCTTCGGCACCGAGAAAGACAAAGTCAACTGTTCATTTTATTTCAAAATTGGAGCATGTC
GTCATGGAGACAGGTGCTCTCGGTTGCACAATAAACCGACGTTTAGCCAGACCATTGCCC
TCTTGAACATTTACCGTAACCCTCAAAACTCTTCCCAGTCTGCTGACGGTTTGCGCTGTG
CCGTGAGCGATGTGGAGATGCAGGAACACTATGATGAGTTTTTTGAGGAGGTTTTTACAG
AAATGGAGGAGAAGTATGGGGAAGTAGAGGAGATGAACGTCTGTGACAACCTGGGAGACC
ACCTGGTGGGGAACGTGTACGTCAAGTTTCGCCGTGAGGAAGATGCGGAAAAGGCTGTGA
TTGACTTGAATAACCGTTGGTTTAATGGACAGCCGATCCACGCCGAGCTGTCACCCGTGA
CGGACTTCAGAGAAGCCTGCTGCCGTCAGTATGAGATGGGAGAATGCACACGAGGCGGCT
TCTGCAACTTCATGCATTTGAAGCCCATTTCCAGAGAGCTGCGGCGGGAGCTGTATGGCC
GCCGTCGCAAGAAGCATAGATCAAGATCCCGATCCCGGGAGCGTCGTTCTCGGTCTAGAG
ACCGTGGTCGTGGCGGTGGCGGTGGCGGTGGTGGAGGTGGCGGCGGACGGGAGCGTGACA
GGAGGCGGTCGAGAGATCGTGAAAGATCTGGGCGATTCTGAGCCATGCCATTTTTACCTT
ATGTCTGCTAGAAAGTGTTGTAGTTGATTGACCAAACCAGTTCATAAGGGGAATTTTTTA
AAAAACAACAAAAAAAAAACATACAAAGATGGGTTTCTGAATAAAAATTTGTAGTGATAA
CAGT
>gi|5803207|gb|NP_006749.1|U2AF1 240aa linear U2 small nuclear
RNA auxillary factor 1; U2 snRNP auxiliary factor small
subunit; splicing factor U2AF 35kDa subunit [Homo sapiens].
MAEYLASIFGTEKDKVNCSFYFKIGACRHGDRCSRLHNKPTFSQTIALLNIYRNPQNSSQ
SADGLRCAVSDVEMQEHYDEFFEEVFTEMEEKYGEVEEMNVCDNLGDHLVGNVYVKFRRE
EDAEKAVIDLNNRWFNGQPIHAELSPVTDFREACCRQYEMGECTRGGFCNFMHLKPISRE

LRRELYGRRRKKHRSRSRSRERRSRSRDRGRGGGGGGGGGGGGRERDRRRSRDRERSGRF
>gi|23308726|gb|NM_003242.3|TGFBR2 2090bp mRNA Homo sapiens transforming growth factor, beta receptor II (70/80kDa) (TGFBR2), mRNA.
GTTGGCGAGGAGTTTCCTGTTTCCCCCGCAGCGCTGAGTTGAAGTTGAGTGAGTCACTCG
CGCGCACGGAGCGACGACACCCCCGCGCGTGCACCCGCTCGGGACAGGAGCCGGACTCCT
GTGCAGCTTCCCTCGGCCGCCGGGGGCCTCCCCGCGCCTCGCCGGCCTCCAGGCCCCTCC
TGGCTGGCGAGCGGGCGCCACATCTGGCCCGCACATCTGCGCTGCCGGCCCGGCGCGGGG
TCCGGAGAGGGCGCGGCGCGGAGCGCAGCCAGGGGTCCGGGAAGGCGCCGTCCGTGCGCT
GGGGGCTCGGTCTATGACGAGCAGCGGGGTCTGCCATGGGTCGGGGGCTGCTCAGGGGCC
TGTGGCCGCTGCACATCGTCCTGTGGACGCGTATCGCCAGCACGATCCCACCGCACGTTC
AGAAGTCGGTTAATAACGACATGATAGTCACTGACAACAACGGTGCAGTCAAGTTTCCAC
AACTGTGTAAATTTTGTGATGTGAGATTTTCCACCTGTGACAACCAGAAATCCTGCATGA
GCAACTGCAGCATCACCTCCATCTGTGAGAAGCCACAGGAAGTCTGTGTGGCTGTATGGA
GAAAGAATGACGAGAACATAACACTAGAGACAGTTTGCCATGACCCCAAGCTCCCCTACC
ATGACTTTATTCTGGAAGATGCTGCTTCTCCAAAGTGCATTATGAAGGAAAAAAAAAAGC
CTGGTGAGACTTTCTTCATGTGTTCCTGTAGCTCTGATGAGTGCAATGACAACATCATCT
TCTCAGAAGAATATAACACCAGCAATCCTGACTTGTTGCTAGTCATATTTCAAGTGACAG
GCATCAGCCTCCTGCCACCACTGGGAGTTGCCATATCTGTCATCATCATCTTCTACTGCT
ACCGCGTTAACCGGCAGCAGAAGCTGAGTTCAACCTGGGAAACCGGCAAGACGCGGAAGC
TCATGGAGTTCAGCGAGCACTGTGCCATCATCCTGGAAGATGACCGCTCTGACATCAGCT
CCACGTGTGCCAACAACATCAACCACAACACAGAGCTGCTGCCCATTGAGCTGGACACCC
TGGTGGGGAAAGGTCGCTTTGCTGAGGTCTATAAGGCCAAGCTGAAGCAGAACACTTCAG
AGCAGTTTGAGACAGTGGCAGTCAAGATCTTTCCCTATGAGGAGTATGCCTCTTGGAAGA
CAGAGAAGGACATCTTCTCAGACATCAATCTGAAGCATGAGAACATACTCCAGTTCCTGA
CGGCTGAGGAGCGGAAGACGGAGTTGGGGAAACAATACTGGCTGATCACCGCCTTCCACG
CCAAGGGCAACCTACAGGAGTACCTGACGCGGCATGTCATCAGCTGGGAGGACCTGCGCA
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GGAGGCCCAAGATGCCCATCGTGCACAGGGACCTCAAGAGCTCCAATATCCTCGTGAAGA
ACGACCTAACCTGCTGCCTGTGTGACTTTGGGCTTTCCCTGCGTCTGGACCCTACTCTGT
CTGTGGATGACCTGGCTAACAGTGGGCAGGTGGGAACTGCAAGATACATGGCTCCAGAAG
TCCTAGAATCCAGGATGAATTTGGAGAATGCTGAGTCCTTCAAGCAGACCGATGTCTACT
CCATGGCTCTGGTGCTCTGGGAAATGACATCTCGCTGTAATGCAGTGGGAGAAGTAAAAG
ATTATGAGCCTCCATTTGGTTCCAAGGTGCGGGAGCACCCCTGTGTCGAAAGCATGAAGG
ACAACGTGTTGAGAGATCGAGGGCGACCAGAAATTCCCAGCTTCTGGCTCAACCACCAGG
GCATCCAGATGGTGTGTGAGACGTTGACTGAGTGCTGGGACCACGACCCAGAGGCCCGTC

TCACAGCCCAGTGTGTGGCAGAACGCTTCAGTGAGCTGGAGCATCTGGACAGGCTCTCGG
GGAGGAGCTGCTCGGAGGAGAAGATTCCTGAAGACGGCTCCCTAAACACTACCAAATAGC
TCTTATGGGGCAGGCTGGGCATGTCCAAAGAGGCTGCCCCTCTCACCAAA
>gi|23308727|gb|NP_003233.3|TGFBR2 567aa linear transforming growth factor, beta receptor II (70/80kDa); transforming growth factor, beta receptor II (70-80kD) [Homo sapiens].
MGRGLLRGLWPLHIVLWTRIASTIPPHVQKSVNNDMIVTDNNGAVKFPQLCKFCDVRFST
CDNQKSCMSNCSITSICEKPQEVCVAVWRKNDENITLETVCHDPKLPYHDFILEDAASPK
CIMKEKKKPGETFFMCSCSSDECNDNIIFSEEYNTSNPDLLLVIFQVTGISLLPPLGVAI
SVIIIFYCYRVNRQQKLSSTWETGKTRKLMEFSEHCAIILEDDRSDISSTCANNINHNTE

LLPIELDTLVGKGRFAEVYKAKLKQNTSEQFETVAVKIFPYEEYASWKTEKDIFSDINLK
HENILQFLTAEERKTELGKQYWLITAFHAKGNLQEYLTRHVISWEDLRKLGSSLARGIAH
LHSDHTPCGRPKMPIVHRDLKSSNILVKNDLTCCLCDFGLSLRLDPTLSVDDLANSGQVG
TARYMAPEVLESRMNLENAESFKQTDVYSMALVLWEMTSRCNAVGEVKDYEPPFGSKVRE
HPCVESMKDNVLRDRGRPEIPSFWLNHQGIQMVCETLTECWDHDPEARLTAQCVAERFSE
LEHLDRLSGRSCSEEKIPEDGSLNTTK
>gi|5174728|gb|NM_006022.1|TSC22 1725bp mRNA Homo sapiens
transforming growth factor beta-stimulated protein TSC-22
(TSC22), mRNA.
CGCCTCTTCACGGCACTGGGATCCGCATCTGCCTGGGATCATCAAGCCCTAGAAGCTGGG
TTTCTTTAAATTAGGGCTGCCGTTTTCTGTTTCTCCCTGGGCTGCGGAAAGCCAGAAGAT
TTTATCTAGCTTATACAAGGCTGCTGGTGTTCCCTCTTTTTTTCCACGAGGGTGTTTTTG
GCTGCAATTGCATGAAATCCCAATGGTGTAGACCAGTGGCGATGGATCTAGGAGTTTACC
AACTGAGACATTTTTCAATTTCTTTCTTGTCATCCTTGCTGGGGACTGAAAACGCTTCTG
TGAGACTTGATAATAGCTCCTCTGGTGCAAGTGTGGTAGCTATTGACAACAAAATCGAGC
AAGCTATGGATCTAGTGAAAAGCCATTTGATGTATGCGGTCAGAGAAGAAGTGGAGGTCC
TCAAAGAGCAAATCAAAGAACTAATAGAGAAAAATTCCCAGCTGGAGCAGGAGAACAATC
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GCTCCCCCCCCTGCCACCACCCAGCCACAGGGCACCACACAGCCCCCCGCCCAGCCAGCAT
CGCAGGGCTCAGGACCAACCGCATAGCTGCCTATGCCCCCGCAGAACTGGCTGCTGCGTG
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CAAGGGTTCTCTCTCCTGCTTGCGCTTAGTGTCCTACATGGGGTTGTGAAGCGATGGAGC
TCCTCACTGGACTCGCCTCTCTCCTCTCCTCCCCCCAGGAGGAACTTGAAAGGAGGGTAA
AAAGACTAAAATGAGGGGGAACAGAGTTCACTGTACAAATTTGACAACTGTCACCAAAAT
TCATAAAAAACAATAGTACTGTGCCTCTTTCTTCTCAAACAATGGATGACACAAAACTAT
GAGAGTGACAAAATGGTGACAGGTAGCTGGGACCTAGGCTATCTTACCATGAAGGTTGTT
TTGCTTATTGTATATTTGTGTATGTAGTGTAACTATTTTGTACAATAGAGGACTGTAACT
ACTATTTAGGTTGTACAGATTGAAATTTAGTTGTTTCATTGGCTGTCTGAGGAGGTGTGG
ACTTTTATATATAGATCTACATAAAAACTGCTACATGACAAAAACCACACCTAAACCCCT
TTTAAGAATTTGGCACAGTTACTCACTTTGTGTAATCTGAAATCTAGCTGCTGAATACGC
TGAAGTAAATCCTTGTTCACTGAAGTCTTTCAATTGAGCTGGTTGAATACTTTGAAAAAT
GCTCAGTTCTAACTAATGAAATGGATTTCCCAGTAGGGGTTTCTGCATATCACCTGTATA
GTAGTTATATGCATATGTTTCTGTGCATGTTCTCTACACAATTGTAAGGTGTCACTGTAT
TTAACTGTTGCACTTGTCAACTTTCAATAAAGCATATAAATGTTG
>gi|5174729|gb|NP_006013.1|TSC22 144aa linear transforming
growth factor beta-stimulated protein TSC-22 [Homo sapiens].
MKSQWCRPVAMDLGVYQLRHFSISFLSSLLGTENASVRLDNSSSGASVVAIDNKIEQAMD
LVKSHLMYAVREEVEVLKEQIKELIEKNSQLEQENNLLKTLASPEQLAQFQAQLQTGSPP
ATTQPQGTTQPPAQPASQGSGPTA
>gi|24432096|gb|NM_152912.2|MTIF3 1693bp mRNA Homo sapiens
mitochondrial translational initiation factor 3 (MTIF3), mRNA.
GCAGATCCGCTGTACTTGCGGGCGCTACAGTATGTCAATCGCTTGCCCCAGCACAGTGGG
CTCCGTGGCTTAAGACTTGAACCAAGTAAACGAAGTTCTCTTACTGAGAAGTCTCAGTTT

CAAAAGAGCTTCTCCTCATCAACTGGGGATGATTACAGTTCTTCCTAAAAAAGCCTACTT
GATGTGAAGACAATGAGGATGAAGACCTTTATGGTGATCCACTTCCACTTAATAGGATGG
CTGCTCTTTTTCTAAAGAGGTTAACACTACAAACTGTAAAGTCTGAAAATAGTTGCATTA
GATGTTTTGGTAAACACATCCTGCAAAAGACAGCACCAGCACAGTTGTCCCCTATTGCTT
CTGCCCCAAGACTCTCCTTCCTAATTCATGCAAAAGCCTTTAGTACCGCTGAAGACACCC
AGAATGAAGGAAAAAAGACAAAAAAGAATAAAACAGCTTTTAGTAACGTTGGAAGAAAAA
TTAGTCAGCGAGTTATTCACTTATTTGATGAGAAGGGCAATGATTTGGGAAACATGCACC
GAGCAAATGTGATTAGACTTATGGATGAGCGAGACCTGCGACTGGTTCAAAGGAACACCA
GCACAGAACCTGCAGAGTATCAGCTCATGACAGGATTGCAGATCCTCCAGGAGCGGCAGA
GGCTGAGGGAGATGGAGAAGGCGAACCCCAAAACTGGACCAACCCTGAGAAAGGAACTGA
TTTTGTCTTCAAATATTGGACAACATGATTTGGACACAAAGACTAAACAGATTCAGCAGT
GGATTAAGAAAAAACACCTAGTCCAGATTACCATAAAGAAAGGAAAAAATGTAGACGTGT
CAGAAAATGAAATGGAGGAGATATTTCATCAAATACTCCAGACTATGCCTGGAATAGCTA
CATTCTCATCTAGGCCACAAGCTGTTCAAGGAGGAAAAGCTTTAATGTGTGTTCTTCGTG
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AAAGCAGTGCAGTTCTTAGCCAAGGGTAAGTACTGCAACTGTCGAGAGCATCTTGTCTTC
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ATGCTAAGCTGAGTCTGTGCCATAGCAAACCATGATATAGCAAGTCTCCAGAATGTGTAC
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>gi|23097266|gb|NP_690876.1|MTIF3 278aa linear mitochondrial translational initiation factor 3 [Homo sapiens].

MAALFLKRLTLQTVKSENSCIRCFGKHILQKTAPAQLSPIASAPRLSFLIHAKAFSTAED
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>gi|27499034|gb|XM_044349.7|CAMK2G 1776bp mRNA Homo sapiens calcium/calmodulin-dependent protein kinase (CaM kinase) II gamma (CAMK2G), mRNA.

CAGCATGGCCACCACCGCCACCTGCACCCGTTTCACCGACGACTACCAGCTCTTCGAGGA
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GTCTGTCTCTGCTGTACCGAGGTGTTTTTTACATTT
>gi|27499035|gb|XP_044349.7|CAMK2G 518aa linear similar to calcium/calmodulin -dependent protein kinase II gamma [Mus musculus] [Homo sapiens].
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>gi|5453881|gb|NM_006213.1|PHKG1 1377bp mRNA Homo sapiens phosphorylase kinase, gamma 1 (muscle) (PHKG1), mRNA.
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GGAGGGGAAGCCATGGAAATACAAGTCAAAGGGGTAAAAAAAAAAAAAAAAAAAAA
>gi|5453882|gb|NP_006204.1|PHKG1 387aa linear phosphorylase kinase, gamma 1 (muscle) [Homo sapiens].
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PEEVRELREATLKEVDILRKVSGHPNIIQLKDTYETNTFFFLVFDLMKRGELFDYLTEKV
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KGQQQNRAALFENTPKAVLLSLAEEDY
>gi|4503412|gb|NM_001945.1|DTR 2360bp mRNA Homo sapiens diphtheria toxin receptor (heparin-binding epidermal growth factor-like growth factor) (DTR), mRNA.
GCTACGCGGGCCACGCTGCTGGCTGGCCTGACCTAGGCGCGCGGGGTCGGGCGGCCGCGC
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ATAAAAAAAAAAAAAAAAAA

>gi|4503413|gb|NP_001936.1|DTR 208aa linear diphtheria toxin receptor (heparin-binding epidermal growth factor-like growth factor); Diphtheria toxin receptor (heparin-binding EGF-like growth factor) [Homo sapiens].

MKLLPSVVLKLFLAAVLSALVTGESLERLRRGLAAGTSNPDPPTVSTDQLLPLGGGRDRK
VRDLQEADLDLLRVTLSSKPQALATPNKEEHGKRKKKGKGLGKKRDPCLRKYKDFCIHGE
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>gi|4507460|gb|NM_003236.1|TGFA 4119bp mRNA Homo sapiens transforming growth factor, alpha (TGFA), mRNA.

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>gi|4507461|gb|NP_003227.1|TGFA 160aa linear transforming growth factor, alpha [Homo sapiens].
MVPSAGQLALFALGIVLAACQALENSTSPLSADPPVAAAVVSHFNDCPDSHTQFCFHGTC
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IHCCQVRKHCEWCRALICRHEKPSALLKGRTACCHSETVV
>gi|6912653|gb|NM_012433.1|SF3B1 4259bp mRNA Homo sapiens splicing factor 3b, subunit 1, 155kDa (SF3B1), mRNA.
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>gi|6912654|gb|NP_036565.1|SF3B1 1304aa linear splicing factor
3b, subunit 1, 155kDa; spliceosome-associated factor 155;
splicing factor 3b, subunit 1, 155kD [Homo sapiens].
MAKIAKTHEDIEAQIREIQGKKAALDEAQGVGLDSTGYYDQEIYGGSDSRFAGYVTSIAA
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>gi|21707321|gb|BC033864.1|BC033864 2321bp mRNA Homo sapiens, Similar to branched chain aminotransferase 1, cytosolic, clone MGC:45234 IMAGE:5186262, mRNA, complete cds.
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>gi|21707322|gb|AAH33864.1|AAH33864 320aa linear Similar to
branched chain aminotransferase 1, cytosolic [Homo sapiens].
MDCSNGCSAECTGEGGSKEVVGTFKAKDLIVTPATILKEKPDPNNLVFGTVFTDHMLTVE
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RSAVRATLPVFDKEELLECIQQLVKLDQEWVPYSTSASLYIRPTFIGTEPSLGVKKPTKA
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>gi|29570794|gb|NM_001895.2|CSNK2A1 2323bp mRNA Homo sapiens
casein kinase 2, alpha 1 polypeptide (CSNK2A1), transcript
variant 2, mRNA.
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>gi|4503095|gb|NP_001886.1|CSNK2A1 391aa linear casein kinase
II alpha 1 subunit isoform a; CK2 catalytic subunit alpha
[Homo sapiens].
MSGPVPSRARVYTDVNTHRPREYWDYESHVVEWGNQDDYQLVRKLGRGKYSEVFEAINIT
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hypothetical protein FLJ14103 (FLJ14103), mRNA.
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>gi|13375964|gb|NP_078965.1|FLJ14103 182aa linear hypothetical protein FLJ14103 [Homo sapiens].

MEPQLGPEAAALRPGWLALLLWVSALSCSFSLPASSLSSLVPQVRTSYNFGRTFLGLDKC
NACIGTSICKKFFKEEIRSDNWLASHLGLPPDSLLSYPANYSDDSKIWRPVEIFRLVSKY
QNEISDRKICASASAPKTCSIERVLRKTERFQKWLQAKRLTPDLVQDCHQGQRELKFLCM
LR

>gi|7658290|gb|AF221842.1|AF221842 3057bp mRNA Homo sapiens U5 snRNP-associated 102 kDa protein mRNA, complete cds.

ACTTTGCTACGGAGTGCATCGGACGTCGAAGCCTAGAGTCTCTGCGTCTTTCCCTCTTCC
GCTGCCTCATTCCTTTCCTTCCTAGCCTTGGTCGTCGCCGCCACCATGAACAAGAAGAAG
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>gi|7658291|gb|AAF66128.1|AAF66128 941aa linear U5 snRNP-associated 102 kDa protein [Homo sapiens].
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>gi|5454165|gb|NM_006370.1|VTI1B 1287bp mRNA Homo sapiens vesicle transport through interaction with t-SNAREs homolog 1B (yeast) (VTI1B), mRNA.

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GCTGCTTTCCATTATCATCTTACTGGAGCTCGCCATCCTGGGAGGCCTGGTTTACTACAA
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TTCAAGGATGCACTGTATAGCCAGGCTGTGGGAGGAGGGAGGAAAGATGAAAAACCACTT
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TATGACTTCTTTAAAAAAAAAAAAAAA

>gi|5454166|gb|NP_006361.1|VTI1B 232aa linear vesicle-associated soluble NSF attachment protein receptor (v-SN; vesicle-associated soluble NSF attachment protein receptor (v-SNARE; homolog of S. cerevisiae VTI1) [Homo sapiens].

MASSAASSEHFEKLHEIFRGLHEDLQGVPERLLGTAGTEEKKKLIRDFDEKQQEANETLA
EMEEELRYAPLSFRNPMMSKLRNYRKDLAKLHREVRSTPLTATPGGRGDMKYGIYAVENE
HMNRLQSQRAMLLQGTESLNRATQSIERSHRIATETDQIGSEIIEELGEQRDQLERTKSR
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>gi|7705992|gb|NM_016440.1|LOC51231 1869bp mRNA Homo sapiens VRK3 for vaccinia related kinase 3 (LOC51231), mRNA.

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CTGTGGAAATTCTTTGCCTGTAGAGGAGCATGTAGGGTCCCAGACCTTTGTCAATCCACA
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CAGTTCTGAGTCTGAAGATACTCTGAGTTCCTCTGAGAGATCCAAAGGCTCCGGGAGCAG
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>gi|7705993|gb|NP_057524.1|LOC51231 474aa linear VRK3 for
vaccinia related kinase 3 [Homo sapiens].
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KKVKWSSTVTSPRLSLFSDGDSSESEDTLSSSERSKGSGSRPPTPKSSPQKTRKSPQVTR
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>gi|27479296|gb|XM_114075.2|TCEA3 1543bp mRNA Homo sapiens
transcription elongation factor A (SII), 3 (TCEA3), mRNA.
CGCCCCCGCCGGGCGTGTGTGTCGTGTGTGTTTGGGGCCCGCGCGGGTTGCGCGCCCTCC
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ACAAGGTGAGGAAGAAGAAAGAGGAAGCGCTGAATTATCTGAACTGGAGAAGCAATAAAA
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>gi|20473950|gb|XP_114075.1|TCEA3 348aa linear similar to Transcription elongation factor A protein 3 (Transcription elongation factor S-II protein 3) (Transcription elongation factor TFIIS.h) [Homo sapiens].

MGQEEELLRIAKKLEKMVARKNTEGALDLLKKLHSCQMSIQLLQTTRIGVAVNGVRKHCS
DKEVVSLAKVLIKNWKRLLDSPGPPKGEKGEEREKAKKKEKGLECSDWKPEAGLSPPRKK
REDPKTRRDSVDSKSSASSSPKRPSVERSNSSKSKAESPKTPSSPLTPTFASSMCLLAPC
YLTGDSVRDKCVEMLSAALKADDDYKDYGVNCDKMASEIEDHIYQELKSTDMKYRNRVRS
RISNLKDPRNPGLRRNVLSGAISAGLIAKMTAEEMASDELRELRNAMTQEAIREHQMAKT
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>gi|21314607|gb|NM_003342.2|UBE2G1 2430bp mRNA Homo sapiens ubiquitin-conjugating enzyme E2G 1 (UBC7 homolog, C. elegans) (UBE2G1), mRNA.

ACCGGCAGCGAGGCGCCGCTCCCGCCGCCTCAGCCCGGCCTTCCTCGGCTCCGGCGCTCC
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AGCCCTTCGGAAGCCCCGGCGCCAGCCCGGGCCCTCGGCAGGGAGGATGACGGAGCTGCA
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CCCAGACCTCTACAAACGTTGTATGTCCTTTTTTAAGCAGAAATAAAATGGTTGAGGACG
AAAAAAAAAAAAAAAAAAAAAAAAAAAAA
>gi|13489085|gb|NP_003333.1|UBE2G1 170aa linear ubiquitin-conjugating enzyme E2G 1 (UBC7 homolog, C. elegans); Ubiquitin-conjugating enzyme E2G (UBC7, C. elegans, human homolog of); ubiquitin-conjugating enzyme E2G (homologous to C. elegans UBC7); ubiquitin-conjugating enzyme E2G 1 (homologous to C. elegans UBC7) [Homo sapiens].
MTELQSALLLRRQLAELNKNPVEGFSAGLIDDNDLYRWEVLIIGPPDTLYEGGVFKAHLT
FPKDYPLRPPKMKFITEIWHPNVDKNGDVCISILHEPGEDKYGYEKPEERWLPIHTVETI
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>gi|21361498|gb|NM_015670.2|SENP3 2258bp mRNA Homo sapiens sentrin/SUMO-specific protease 3 (SENP3), mRNA.
GAAGCTTGAGGCCGGAGACGCCCGCCTTCGGGCCCGTCCGCCCGGCTTCCCCGCTCCCGG
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TGGGCCCCCCATGGAGGAAGATGGACTCAGGTGGACTCCAAAGTCTCCTCTGGACCCTGA
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>gi|21361499|gb|NP_056485.2|SENP3 574aa linear sentrin/SUMO-specific protease 3 [Homo sapiens].

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KVHFFNSFFYDKLRTKGYDGVKRWTKNVDIFNKELLLIPIHLEVHWSLISVDVRRRTITY
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KHLALSQPFSFTQQDMPKLRRQIYKELCHCKLTV

>gi|5803166|gb|NM_006802.1|SF3A3 2733bp mRNA Homo sapiens splicing factor 3a, subunit 3, 60kDa (SF3A3), mRNA.

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TCATGGACGTCATGGCTAAAGAGATGCTCACCAAGAAGTCCACGCTCCGGGACCAGATCA
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GGGATTTGTATGATGATAAGGATGGATTACGAAAGGAGGAGCTCAATGCCATTTCAGGAC
CCAATGAGTTTGCTGAATTCTATAATAGACTCAAGCAAATAAAGGAATTCCACCGGAAGC
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>gi|5803167|gb|NP_006793.1|SF3A3 501aa linear splicing factor 3a, subunit 3, 60kDa; pre-mRNA splicing factor SF3a (60kD) [Homo sapiens].

METILEQQRRYHEEKERLMDVMAKEMLTKKSTLRDQINSDHRTRAMQDRYMEVSGNLRDL
YDDKDGLRKEELNAISGPNEFAEFYNRLKQIKEFHRKHPNEICVPMSVEFEELLKARENP
SEEAQNLVEFTDEEGYGRYLDLHDCYLKYINLKASEKLDYITYLSIFDQLFDIPKERKNA
EYKRYLEMLLEYLQDYTDRVKPLQDQNELFGKIQAEFEKKWENGTFPGWPKETSSALTHA
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EEQISESESEDEENEIIYNPKNLPLGWDGKPIPYWLYKLHGLNINYNCEICGNYTYRGPK
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>gi|28882054|gb|NM_005011.2|NRF1 2514bp mRNA Homo sapiens nuclear respiratory factor 1 (NRF1), mRNA.

GAGGCTGCGAGGAGCCGGCGCGGTCGCAGTCTCCACGGCGCAGGCCCACGGTAGCGCAGC
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GTCGCCACCCTGGCTGAGGCCACCTTACAAGGTGGGGGACAGATCGTCTTGTCTGGGGAA
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TGGGTCAGAGATGTGCTTGCAAGGTCCAGGGACTGCGTGGTCTGCCAGCTGAGATGCTCC
TCGGGCTGGCCCAGGTGCTGACCTTGCCACAGGCAGATGAATGTCTTGAAAGCTCCCGGG
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>gi|28882055|gb|NP_005002.2|NRF1 522aa linear nuclear respiratory factor 1 [Homo sapiens].

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EYTTRVGQQAIVLCISPSKPNPVFKVFGAAPLENVVRKYKSMILEDLESALAEHAPAPQE
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>gi|6996000|gb|NM_001663.2|ARF6 1806bp mRNA Homo sapiens ADP-ribosylation factor 6 (ARF6), mRNA.
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GAGGAAGGCGGAGGAGCGGGAACCGCGGCGGCGCTCGCGCGGCGCCTGCGGGGGGAAGGG
CAGTTCCGGGCCGGGCCGCGCCTCAGCAGGGCGGCGGCTCCCAGCGCAGTCTCAGGGCCC
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CAAATAATGAGTAATATTGTAATATGTTCCAGTTGCACCTCAGTATGTTAAACAGGTAAT
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ATAAAC
>gi|4502211|gb|NP_001654.1|ARF6 175aa linear ADP-ribosylation factor 6 [Homo sapiens].
MGKVLSKIFGNKEMRILMLGLDAAGKTTILYKLKLGQSVTTIPTVGFNVETVTYKNVKFN
VWDVGGQDKIRPLWRHYYTGTQGLIFVVDCADRDRIDEARQELHRIINDREMRDAIILIF
ANKQDLPDAMKPHEIQEKLGLTRIRDRNWYVQPSCATSGDGLYEGLTWLTSNYKS
>gi|23510442|gb|NM_003809.2|TNFSF12 1407bp mRNA Homo sapiens tumor necrosis factor (ligand) superfamily, member 12 (TNFSF12), transcript variant 1, mRNA.
CTCTCCCCGGCCCGATCCGCCCGCCGGCTCCCCCTCCCCCGATCCCTCGGGTCCCGGGAT
GGGGGGGCGGTGAGGCAGGCACAGCCCCCCGCCCCCATGGCCGCCCGTCGGAGCCAGAGG
CGGAGGGGGCGCCGGGGGGAGCCGGGCACCGCCCTGCTGGTCCCGCTCGCGCTGGGCCTG

GGCCTGGCGCTGGCCTGCCTCGGCCTCCTGCTGGCCGTGGTCAGTTTGGGGAGCCGGGCA
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GTCATAAAAAAAAAAAAAAAAAAAAA

>gi|4507597|gb|NP_003800.1|TNFSF12 249aa linear tumor necrosis factor (ligand) superfamily, member 12 isoform 1 precursor; APO3/DR3 ligand; TNF-related WEAK inducer of apoptosis [Homo sapiens].

MAARRSQRRRGRRGEPGTALLVPLALGLGLALACLGLLLAVVSLGSRASLSAQEPAQEEL
VAEEDQDPSELNPQTEESQDPAPFLNRLVRPRRSAPKGRKTRARRAIAAHYEVHPRPGQD
GAQAGVDGTVSGWEEARINSSSPLRYNRQIGEFIVTRAGLYYLYCQVHFDEGKAVYLKLD
LLVDGVLALRCLEEFSATAASSLGPQLRLCQVSGLLALRPGSSLRIRTLPWAHLKAAPFL
TYFGLFQVH

>gi|11496238|gb|NM_021975.1|RELA 2444bp mRNA Homo sapiens v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian) (RELA), mRNA.

GGCACGAGGCGGGGCCGGGTCGCAGCTGGGCCCGCGGCATGGACGAACTGTTCCCCCTCA
TCTTCCCGGCAGAGCAGCCCAAGCAGCGGGGCATGCGCTTCCGCTACAAGTGCGAGGGGC
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TCAAGATCAATGGCTACACAGGACCAGGGACAGTGCGCATCTCCCTGGTCACCAAGGACC
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>gi|11496239|gb|NP_068810.1|RELA 537aa linear v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65; v-rel avian reticuloendotheliosis viral oncogene homolog A (nuclear factor of kappa light polypeptide gene enhancer in B-cells 3 (p65)) [Homo sapiens].

MDELFPLIFPAEQPKQRGMRFRYKCEGRSAGSIPGERSTDTTKTHPTIKINGYTGPGTVR
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IQTNNNPFQVPIEEQRGDYDLNAVRLCFQVTVRDPSGRPLRLPPVLSHPIFDNRAPNTAE
LKICRVNRNSGSCLGGDEIFLLCDKVQKEDIEVYFTGPGWEARGSFSQADVHRQVAIVFR
TPPYADPSLQAPVRVSMQLRRPSDRELSEPMEFQYLPDTDDRHRIEEKRKRTYETFKSIM
KKSPFSGPTDPRPPPRRIAVPSRSSASVPKPAPQPYPFTSSLSTINYDEFPTMVFPSGQI
SQASALAPAPPQVLPQAPAPAPAPAMVSALAQAPAPVPVLAPGPPQAVAPPAPKPTQAGE
GTLSEALLQLQFDDEDLGALLGNSTDPAVFTDLASVDNSEFQQLLNQGIPVAPHTTEPML
MEYPEAITRLVTAQRPPDPAPAPLGAPGLPNGLLSGDEDFSSIADMDFSALLSQISS

>gi|23312372|gb|NM_001065.2|TNFRSF1A 2236bp mRNA Homo sapiens tumor necrosis factor receptor superfamily, member 1A (TNFRSF1A), mRNA.

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ATTTTCTCAGAATTCTCTGGACTGAGGCTCCAGTTCTGGCCTTTGGGGTTCAAGATCACT
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CTGAGCCCAAATGGGGGAGTGAGAGGCCATAGCTGTCTGGCATGGGCCTCTCCACCGTGC
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TTGTACATACACTAAAATTCTGAAGTTAAAGCTCTGCTCTTGGAAAAAAAAAAAAAAAAA
AAAAAAAAAAAAAAA

>gi|4507575|gb|NP_001056.1|TNFRSF1A 455aa linear tumor necrosis factor receptor 1 precursor; tumor necrosis factor receptor type 1; tumor necrosis factor-alpha receptor; tumor necrosis factor binding protein 1 [Homo sapiens].

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SCSNCKKSLECTKLCLPQIENVKGTEDSGTTVLLPLVIFFGLCLLSLLFIGLMYRYQRWK
SKLYSIVCGKSTPEKEGELEGTTTKPLAPNPSFSPTPGFTPTLGFSPVPSSTFTSSSTYT
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AVVENVPPLRWKEFVRRLGLSDHEIDRLELQNGRCLREAQYSMLATWRRRTPRREATLEL
LGRVLRDMDLLGCLEDIEEALCGPAALPPAPSLLR
>gi|4506738|gb|NM_003952.1|RPS6KB2 1735bp mRNA Homo sapiens
ribosomal protein S6 kinase, 70kDa, polypeptide 2 (RPS6KB2),
mRNA.
AGAGACTCGTGCCGAATGGCACGAGGCCGACGGGCCCGCGGGGCCGGCGCCGCCATGGCG
GCCGTGTTTGATTTGGATTTGGAGACGGAGGAAGGCAGCGAGGGCGAGGGCGAGCCAGAG
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>gi|4506739|gb|NP_003943.1|RPS6KB2 495aa linear ribosomal
protein S6 kinase, 70kDa, polypeptide 2; ribosomal protein S6
kinase, 70kD, polypeptide 2; p70 ribosomal S6 kinase beta
[Homo sapiens].
MARGRRARGAGAAMAAVFDLDLETEEGSEGEGEPELSPADACPLAELRAAGLEPVGHYEE
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LPPYLTPDARDLVKKFLKRNPSQRIGGGPGDAADVQRHPFFRHMNWDDLLAWRVDPPFRP
CLQSEEDVSQFDTRFTRQTPVDSPDDTALSESANQAFLGFTYVAPSVLDSIKEGFSFQPK
LRSPRRLNSSPRVPVSPLKFSPFEGFRPSPSLPEPTELPLPPLLPPPPPSTTAPLPIRPP
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>gi|11995473|gb|NM_019884.1|GSK3A 2169bp mRNA Homo sapiens glycogen synthase kinase 3 alpha (GSK3A), mRNA.

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GCTGGGGCAGCCCGGGCAGCCCGAGCCCCGCAGCCTGGGCCTGTGCTCGGCGCCATGAGC
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>gi|11995474|gb|NP_063937.1|GSK3A 483aa linear glycogen synthase kinase 3 alpha [Homo sapiens].

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NSS
>gi|7019350|gb|NM_013246.1|CLC 1689bp mRNA Homo sapiens
cardiotrophin-like cytokine (CLC), mRNA.
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>gi|7019351|gb|NP_037378.1|CLC 225aa linear cardiotrophin-like
cytokine; neurotrophin-1/B-cell stimulating factor-3 [Homo
sapiens].
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zinc finger protein 213 (ZNF213), mRNA.
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CTAACGCCCTGCT
>gi|14777854|gb|XP_036493.1|ZNF213 459aa linear similar to Zinc finger protein 213 (Putative transcription factor CR53) [Homo sapiens].
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>gi|21536281|gb|NM_003656.3|CAMK1 1501bp mRNA Homo sapiens calcium/calmodulin-dependent protein kinase I (CAMK1), mRNA.
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A
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>gi|13186237|gb|NM_023107.1|FGFR1 2590bp mRNA Homo sapiens
fibroblast growth factor receptor 1 (fms-related tyrosine
kinase 2, Pfeiffer syndrome) (FGFR1), transcript variant 5,
mRNA.
CCTCTTGCGGCCACAGGCGCGGCGTCCTCGGCGGCGGGCGGCAGCTAGCGGGAGCCGGGA
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AAAAAAAAAA
>gi|13186238|gb|NP_075595.1|FGFR1 302aa linear fibroblast growth factor receptor 1 isoform 5 precursor; fms-related tyrosine kinase-2; heparin-binding growth factor receptor; FMS-like tyrosine kinase 2; basic fibroblast growth factor receptor 1; N-sam tyrosine kinase; FLG protein; protein-tyrosine kinase; tyrosylprotein kinase; hydroxyaryl-protein kinase [Homo sapiens].
MWSWKCLLFWAVLVTATLCTARPSPTLPEQDALPSSEDDDDDDDSSSEEKETDNTKPNRM
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>gi|4758007|gb|NM_004071.1|CLK1 1834bp mRNA Homo sapiens CDC-like kinase 1 (CLK1), mRNA.
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>gi|4758008|gb|NP_004062.1|CLK1 484aa linear CDC-like kinase
1; protein tyrosine kinase STY [Homo sapiens].
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KKSI
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MUS81 endonuclease (MUS81), mRNA.
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endothelial growth factor [Homo sapiens].
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>gi|16306546|gb|NP_387498.1|FGF18 207aa linear fibroblast growth factor 18 precursor [Homo sapiens].
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factor-regulated tyrosine kinase substrate [Homo sapiens].
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>gi|10863937|gb|NP_066960.1|TNFAIP1 316aa linear tumor
necrosis factor, alpha-induced protein 1 [Homo sapiens].
MSGDTCLCPASGAKPKLSGFKGGGLGNKYVQLNVGGSLYYTTVRALTRHDTMLKAMFSGR
MEVLTDKEGWILIDRCGKHFGTILNYLRDDTITLPQNRQEIKELMAEAKYYLIQGLVNMC
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>gi|27597077|gb|NM_006293.2|TYRO3 3949bp mRNA Homo sapiens
TYRO3 protein tyrosine kinase (TYRO3), mRNA.
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>gi|27597078|gb|NP_006284.2|TYRO3 890aa linear TYRO3 protein tyrosine kinase; Brt; Dtk; Sky; Tif; Tyro3 protein tyrosine kinase (sea-related receptor tyrosine kinase); tyrosine-protein kinase receptor TYRO3 precursor [Homo sapiens].

MALRRSMGRPGLPPLPLPPPPRLGLLLAALASLLLPESAAAGLKLMGAPVKLTVSQGQPV
KLNCSVEGMEEPDIQWVKDGAVVQNLDQLYIPVSEQHWIGFLSLKSVERSDAGRYWCQVE
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>gi|4502884|gb|NM_003992.1|CLK3 1762bp mRNA Homo sapiens CDC-
like kinase 3 (CLK3), transcript variant phclk3, mRNA.
TGGGGCACTGGTACCTCCAGGACCTGGAGTGTACTGGAAGAAATGGTGCAGTCCAGATGC
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>gi|4502885|gb|NP_003983.1|CLK3 490aa linear CDC-like kinase 3
isoform hclk3 [Homo sapiens].
MHHCKRYRSPEPDPYLSYRWKRRRSYSREHEGRLRYPSRREPPPRRSRSRSHDRLPYQRR
YRERRDSDTYRCEERSPSFGEDYYGPSRSRHRRRSRERGPYRTRKHAHHCHKRRTRSCSS
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SQVALKIIRNVGKYREAARLEINVLKKIKEKDKENKFLCVLMSDWFNFHGHMCIAFELLG

KNTFEFLKENNFQPYPLPHVRHMAYQLCHALRFLHENQLTHTDLKPENILFVNSEFETLY
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GCILFEYYRGFTLFQTHENREHLVMMEKILGPIPSHMIHRTRKQKYFYKGGLVWDENSSD
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SFHTSRNPSR

>gi|9910121|gb|NM_020249.1|ADAMTS9 3674bp mRNA Homo sapiens a disintegrin-like and metalloprotease (reprolysin type) with thrombospondin type 1 motif, 9 (ADAMTS9), mRNA.

GCGGGAAGCACCATGCAGTTTGTATCCTGGGCCACACTGCTAACGCTCCTGGTGCGGGAC
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>gi|9910122|gb|NP_064634.1|ADAMTS9 1072aa linear a disintegrin and metalloproteinase with thrombospondin motifs-9 preproprotein [Homo sapiens].

MQFVSWATLLTLLVRDLAEMGSPDAAAAVRKDRLHPRQVKLLETLGEYEIVSPIRVNALG
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GDYFIEPLQSMDEQEDEEEQNKPHIIYRRSAPQREPSTGRHACDTSEHKNRHSKDKKKTR
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KLVCTRESDQLTVSDQRCDRLPQPGHITEPCGTDCDLRWHVASRSECSAQCGLGYRTLDI
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>gi|17981697|gb|NM_001262.2|CDKN2C 2104bp mRNA Homo sapiens cyclin-dependent kinase inhibitor 2C (p18, inhibits CDK4) (CDKN2C), transcript variant 1, mRNA.

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AAAA

>gi|4502751|gb|NP_001253.1|CDKN2C 168aa linear cyclin-dependent kinase inhibitor 2C; cyclin-dependent kinase 6 inhibitor p18; cyclin-dependent kinase 4 inhibitor C; cyclin-dependent inhibitor; CDK6 inhibitor p18 [Homo sapiens].

MAEPWGNELASAAARGDLEQLTSLLQNNVNVNAQNGFGRTALQVMKLGNPEIARRLLLRG
ANPDLKDRTGFAVIHDAARAGFLDTLQTLLEFQADVNIEDNEGNLPLHLAAKEGHLRVVE
FLVKHTASNVGHRNHKGDTACDLARLYGRNEVVSLMQANGAGGATNLQ

>gi|23510344|gb|NM_002037.3|FYN 2650bp mRNA Homo sapiens FYN oncogene related to SRC, FGR, YES (FYN), transcript variant 1, mRNA.

GCCGCGCTGGTGGCGGCGGCGCGTCGTTGCAGTTGCGCCATCTGTCAGGAGCGGAGCCGG

CGAGGAGGGGGCTGCCGCGGGCGAGGAGGAGGGGTCGCCGCGAGCCGAAGGCCTTCGAGA
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>gi|4503823|gb|NP_002028.1|FYN 537aa linear protein-tyrosine kinase fyn isoform a; proto-oncogene tyrosine-protein kinase fyn; src/yes-related novel gene; src-like kinase; c-syn

protooncogene; tyrosine kinase p59fyn(T); OKT3-induced calcium influx regulator [Homo sapiens].
MGCVQCKDKEATKLTEERDGSLNQSSGYRYGTDPTPQHYPSFGVTSIPNYNNFHAAGGQG
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>gi|15055546|gb|NM_000800.2|FGF1 2357bp mRNA Homo sapiens fibroblast growth factor 1 (acidic) (FGF1), transcript variant 1, mRNA.
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AAAAAAAAAAAAAAAAA
>gi|4503697|gb|NP_000791.1|FGF1 155aa linear fibroblast growth factor 1 (acidic) isoform 1 precursor; heparin-binding growth factor 1 precursor; endothelial cell growth factor, alpha; endothelial cell growth factor, beta [Homo sapiens].
MAEGEITTFTALTEKFNLPPGNYKKPKLLYCSNGGHFLRILPDGTVDGTRDRSDQHIQLQ
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>gi|27552761|gb|NM_002825.3|PTN 1029bp mRNA Homo sapiens pleiotrophin (heparin binding growth factor 8, neurite growth-promoting factor 1) (PTN), mRNA.
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>gi|4506281|gb|NP_002816.1|PTN 168aa linear pleiotrophin (heparin binding growth factor 8, neurite growth-promoting factor 1); heparin affin regulatory protein; heparin-binding growth-associated molecule [Homo sapiens].
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>gi|4504009|gb|NP_000160.1|GLA 429aa linear galactosidase, alpha [Homo sapiens].
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ANYVHSKGLKLGIYADVGNKTCAGFPGSFGYYDIDAQTFADWGVDLLKFDGCYCDSLENL
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MQMSLKDLL
>gi|18587778|gb|XM_091624.1|LOC162542 287bp mRNA Homo sapiens similar to ADP-ribosylation factor 1 (LOC162542), mRNA.
GTCTGATTTTTATGGTTGACAGTAATGACAGAGAGCAGATTGATGAGGCCTGGGAAGTGC
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>gi|18587779|gb|XP_091624.1|LOC162542 91aa linear similar to ADP-ribosylation factor 1 [Homo sapiens].
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T

>gi|4557573|gb|NP_000392.1|EXT2 718aa linear exostoses (multiple) 2 [Homo sapiens].

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>gi|27597083|gb|NM_006838.2|METAP2 1908bp mRNA Homo sapiens methionyl aminopeptidase 2 (METAP2), mRNA.

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[Homo sapiens].

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choline/ethanolamine kinase isoform a [Homo sapiens].

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>gi|4757756|gb|NP_004030.1|ANXA2 339aa linear annexin A2; annexin II; annexin II (lipocortin II); calpactin I, heavy polypeptide (p36); lipocortin II; Annexin II (lipocortin I); annexin II (lipocortin II; calpactin I, heavy polypeptide) [Homo sapiens].

MSTVHEILCKLSLEGDHSTPPSAYGSVKAYTNFDAERDALNIETAIKTKGVDEVTIVNIL
TNRSNAQRQDIAFAYQRRTKKELASALKSALSGHLETVILGLLKTPAQYDASELKASMKG
LGTDEDSLIEIICSRTNQELQEINRVYKEMYKTDLEKDIISDTSGDFRKLMVALAKGRRA
EDGSVIDYELIDQDARDLYDAGVKRKGTDVPKWISIMTERSVPHLQKVFDRYKSYSPYDM
LESIRKEVKGDLENAFLNLVQCIQNKPLYFADRLYDSMKGKGTRDKVLIRIMVSRSEVDM
LKIRSEFKRKYGKSLYYYIQQDTKGDYQKALLYLCGGDD

>gi|27484939|gb|XM_084635.3|LOC143785 1982bp mRNA Homo sapiens similar to hypothetical protein XP_084635 [Homo sapiens] (LOC143785), mRNA.

TACTATCAGGGGGCAAGAGCCTTTCTCTCCAGCTACACACTCCATCTCCCGGGAGCAAGG
GGAAACTCCGAGAGGAGGGCAACAGAGCCAGCATCTTGCCAGGGCCCCGGAGGAGGGGTT
CCCCGCTACGCCTGTGCCGGAGGAGTTCCAGTCACCGAGCGAGGGGCGCAAGGGTGGGTG
CATCCTGCGCTGCGGCGGGCGCGCTACCCAGACGCTGGTGTGCAGAGCCACATGAAGCCT
GCTGGGGACTGGGGGCCAGGGAGCAGCAAGCCAGCTGGGACTGAGGCGGACGCTGTCTCA
GGGAGACGCTGACTCGCAAAGACACTCCCTTCCTTGTGCCTGGGTAAAAAGTCTCCTCCT
GGGGTCCCTGGCCATCCTGAATATCCAGAATGGTGTTTCTGAAGTTCTTCTGCATGAGTT
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TGGAGGATGCTGGGCGCGTGCTGGAGGGCATCAGCAAAAGCATCTCCTACGACCTAGACG
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ACTTTGTTTTTAGTTTTGTTTTTGTACATTATTTATGTGATTGTTATGGAATTGTCACCT
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TGCAGATTATGTGGAAATACCCAAAGATAATAGTGCATAGCTCCTTTCAGCCTCTAGCCT
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CTTTCTCATTGTATGTCTATGCTTTGTCATCAGTGCTATAGTAAATTACAAAGAAATAGG
TAGATTGTATGAACATACCCACAAATGCCTATGATTTAGGTTACCAATGTATTCTTTCTC
ATTTGGGGTTTTGCTTCTGTCTGTCTGTTTATTGGAAACTTGTACTTCAAGTAGGGGGAA
TCCTAATTCTAATAACTCCTTAGCTAAGTTTTATTATTCAGGCAATAAACATGTTTTCAT
GT

>gi|18578340|gb|XP_084635.1|LOC143785 211aa linear similar to hypothetical protein XP_084635 [Homo sapiens].

MVFLKFFCMSFFCHLCQGYFDGPLYPEMSNGTLHHYFVPDGDYEENDDPEKCQLLFRVSD
HRRCSQGEGSQVGSLLSLTLREEFTVLGRQVEDAGRVLEGISKSISYDLDGEESYGKYLR
RESHQIGDAYSNSDKSLTELESKFKQGQEQDSRQESRLNEDFLGMLVHTRSLLKETLDIS
VGLRDKYELLALTIRSHGTRLGRLKNDYLKV

>gi|4507464|gb|NM_003239.1|TGFB3 2574bp mRNA Homo sapiens transforming growth factor, beta 3 (TGFB3), mRNA.

CCTGTTTAGACACATGGACAACAATCCCAGCGCTACAAGGCACACAGTCCGCTTCTTCGT
CCTCAGGGTTGCCAGCGCTTCCTGGAAGTCCTGAAGCTCTCGCAGTGCAGTGAGTTCATG
CACCTTCTTGCCAAGCCTCAGTCTTTGGGATCTGGGGAGGCCGCCTGGTTTTCCTCCCTC
CTTCTGCACGTCTGCTGGGGTCTCTTCCTCTCCAGGCCTTGCCGTCCCCCTGGCCTCTCT
TCCCAGCTCACACATGAAGATGCACTTGCAAAGGGCTCTGGTGGTCCTGGCCCTGCTGAA
CTTTGCCACGGTCAGCCTCTCTCTGTCCACTTGCACCACCTTGGACTTCGGCCACATCAA
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CCCTGAGCCAACGGTGATGACCCACGTCCCCTATCAGGTCCTGGCCCTTTACAACAGCAC
CCGGGAGCTGCTGGAGGAGATGCATGGGGAGAGGGAGGAAGGCTGCACCCAGGAAAACAC
CGAGTCGGAATACTATGCCAAAGAAATCCATAAATTCGACATGATCCAGGGGCTGGCGGA
GCACAACGAACTGGCTGTCTGCCCTAAAGGAATTACCTCCAAGGTTTTCCGCTTCAATGT
GTCCTCAGTGGAGAAAAATAGAACCAACCTATTCCGAGCAGAATTCCGGGTCTTGCGGGT
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GTGCGACAGAGAGAGGGGAGAGAGAACCACCACTGCCTGACTGCCCGCTCCTCGGGAAAC
ACACAAGCAACAAACCTCACTGAGAGGCCTGGAGCCCACAACCTTCGGCTCCGGGCAAAT
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CTGTGACGCAGACAGAGGGGATGGGGATAGAGGAAAGGGATGGTAAGTTGAGATGTTGTG
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ACGAAGACAAAGTCCCAGAATTGTATCTCATACTGTCTGGGATTAAGGGCAAATCTATTA
CTTTTGCAAACTGTCCTCTACATCAATTAACATCGTGGGTCACTACAGGGAGAAAATCCA
GGTCATGCAGTTCCTGGCCCATCAACTGTATTGGGCCTTTTGGATATGCTGAACGCAGAA
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TCGATCATATTTCCCCTTGGACACTTGGTTAGACGCCTTCCAGGTCAGGATGCACATTTC
TGGATTGTGGTTCCATGCAGCCTTGGGGCATTATGGGTCTTCCCCCACTTCCCCTCCAAG
ACCCTGTGTTCATTTGGTGTTCCTGGAAGCAGGTGCTACAACATGTGAGGCATTCGGGGA
AGCTGCACATGTGCCACACAGTGACTTGGCCCCAGACGCATAGACTGAGGTATAAAGACA
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>gi|4507465|gb|NP_003230.1|TGFB3 412aa linear transforming growth factor, beta 3 [Homo sapiens].

MKMHLQRALVVLALLNFATVSLSLSTCTTLDFGHIKKKRVEAIRGQILSKLRLTSPPEPT
VMTHVPYQVLALYNSTRELLEEMHGEREEGCTQENTESEYYAKEIHKFDMIQGLAEHNEL
AVCPKGITSKVFRFNVSSVEKNRTNLFRAEFRVLRVPNPSSKRNEQRIELFQILRPDEHI
AKQRYIGGKNLPTRGTAEWLSFDVTDTVREWLLRRESNLGLEISIHCPCHTFQPNGDILE
NIHEVMEIKFKGVDNEDDHGRGDLGRLKKQKDHHNPHLILMMIPPHRLDNPGQGGQRKKR
ALDTNYCFRNLEENCCVRPLYIDFRQDLGWKWVHEPKGYYANFCSGPCPYLRSADTTHST
VLGLYNTLNPEASASPCCVPQDLEPLTILYYVGRTPKVEQLSNMVVKSCKCS

>gi|21735553|gb|NM_002419.2|MAP3K11 3603bp mRNA Homo sapiens mitogen-activated protein kinase kinase kinase 11 (MAP3K11), mRNA.

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CAGGAACAGGCAGTCTCGGCCCAACTGCGGACGCTCCCTCCACCCCCTGCGCAAAAAGAC
CCAACCGGAGTTGAGGCGCTGCCCCTGAAGGCCCCACCTTACACTTGGCGGGGGCCGGAG
CCAGGCTCCCAGGACTGCTCCAGAACCGAGGGAAGCTCGGGTCCCTCCAAGCTAGCCATG
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CAGCTGCCTCAGTTTCCCCAGGGACCCCACCCCCCTTTGGGGGTCAGGAACACTACACTG
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CCCCCAGCTCACCTGCCCTACTGGGGCCCAACACTGTACCCAGCTGGTTGGGAGGACCAG
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CCTAGGGCTTAGAGCATGGACCTCCTGCCCTGGGGGTCATCTGGGGCCAGGGCTCTCTGG
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AAGCCAAATAAACTCAAAAGCTGTCTCCCCAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
AAA

>gi|4505195|gb|NP_002410.1|MAP3K11 847aa linear mitogen-activated protein kinase kinase kinase 11; mixed lineage kinase 3; SH3 domain-containing proline-rich kinase; protein-tyrosine kinase PTK1 [Homo sapiens].

MEPLKSLFLKSPLGSWNGSGSGGGGGGGGRPEGSPKAAGYANPVWTALFDYEPSGQDEL
ALRKGDRVEVLSRDAAISGDEGWWAGQVGGQVGIFPSNYVSRGGGPPPCEVASFQELRLE
EVIGIGGFGKVYRGSWRGELVAVKAARQDPDEDISVTAESVRQEARLFAMLAHPNIIALK
AVCLEEPNLCLVMEYAAGGPLSRALAGRRVPPHVLVNWAVQIARGMHYLHCEALVPVIHR
DLKSNNILLLQPIESDDMEHKTLKITDFGLAREWHKTTQMSAAGTYAWMAPEVIKASTFS
KGSDVWSFGVLLWELLTGEVPYRGIDCLAVAYGVAVNKLTLPIPSTCPEPFAQLMADCWA
QDPHRRPDFASILQQLEALEAQVLREMPRDSFHSMQEGWKREIQGLFDELRAKEKELLSR
EEELTRAAREQRSQAEQLRRREHLLAQWELEVFERELTLLLQQVDRERPHVRRRRGTFKR
SKLRARDGGERISMPLDFKHRITVQASPGLDRRRNVFEVGPGDSPTFPRFRAIQLEPAEP
GQAWGRQSPRRLEDSSNGERRACWAWGPSSPKPGEAQNGRRRSRMDEATWYLDSDDSSPL
GSPSTPPALNGNPPRPSLEPEEPKRPVPAERGSSSGTPKLIQRALLRGTALLASLGLGRD
LQPPGGPGRERGESPTTPPTPTPAPCPTEPPPSPLICFSLKTPDSPPTPAPLLLDLGIPV
GQRSAKSPRREEEPRGGTVSPPPGTSRSAPGTPGTPRSPPLGLISRPRPSPLRSRIDPWS
FVSAGPRPSPLPSPQPAPRRAPWTLFPDSDPFWDSPPANPFQGGPQDCRAQTKDMGAQAP
WVPEAGP

>gi|4505784|gb|NM_000294.1|PHKG2 1571bp mRNA Homo sapiens phosphorylase kinase, gamma 2 (testis) (PHKG2), mRNA.

AAGGTGAGCGACTGCAGGCAAACCCGGCGACAGCGCAGCTCGCGTCGACCCTGGCTCCTC
TGCCTGCCCCCTCAGGCCCCCGCCTCCTTCAGGATGACGCTGGACGTGGGGCCGGAGGAT
GAGCTGCCCGACTGGGCCGCCGCCAAAGAGTTTTACCAGAAGTACGACCCTAAGGACGTC
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ACCAGGTCCATCATGCGGTCTCTGCTGGAAGCAGTGAGCTTTCTCCATGCCAACAACATT
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ACACGCCAGGG

>gi|4505785|gb|NP_000285.1|PHKG2 406aa linear phosphorylase kinase, gamma 2 (testis); Phosphorylase kinase, gamma 2 (testis/liver) [Homo sapiens].

MTLDVGPEDELPDWAAAKEFYQKYDPKDVIGRGVSSVVRRCVHRATGHEFAVKIMEVTAE
RLSPEQLEEVREATRRETHILRQVAGHPHIITLIDSYESSSFMFLVFDLMRKGELFDYLT
EKVALSEKETRSIMRSLLEAVSFLHANNIVHRDLKPENILLDDNMQIRLSDFGFSCHLEP
GEKLRELCGTPGYLAPEILKCSMDETHPGYGKEVDLWACGVILFTLLAGSPPFWHRRQIL
MLRMIMEGQYQFSSPEWDDRSSTVKDLISRLLQVDPEARLTAEQALQHPFFERCEGSQPW
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WVKKGEQQNRAALFQHRPPGPFPIMGPEEEGDSAAITEDEAVLVLG

>gi|5453789|gb|NM_006169.1|NNMT 952bp mRNA Homo sapiens nicotinamide N-methyltransferase (NNMT), mRNA.

TGAACTCTGGATGCTGTTAGCCTGAGACTCAGGAAGACAACTTCTGCAGGGTCACTCCCT
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>gi|5453790|gb|NP_006160.1|NNMT 264aa linear nicotinamide N-methyltransferase [Homo sapiens].

MESGFTSKDTYLSHFNPRDYLEKYYKFGSRHSAESQILKHLLKNLFKIFCLDGVKGDLLI
DIGSGPTIYQLLSACESFKEIVVTDYSDQNLQELEKWLKKEPEAFDWSPVVTYVCDLEGN
RVKGPEKEEKLRQAVKQVLKCDVTQSQPLGAVPLPPADCVLSTLCLDAACPDLPTYCRAL
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SYSSTMANNEGLFSLVARKLSRPL

>gi|4507668|gb|NM_003295.1|TPT1 830bp mRNA Homo sapiens tumor protein, translationally-controlled 1 (TPT1), mRNA.

CCCCCCCGAGCGCCGCTCCGGCTGCACCGCGCTCGCTCCGAGTTTCAGGCTCGTGCTAAG
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>gi|4507669|gb|NP_003286.1|TPT1 172aa linear tumor protein, translationally-controlled 1; fortilin; histamine-releasing factor [Homo sapiens].
MIIYRDLISHDEMFSDIYKIREIADGLCLEVEGKMVSRTEGNIDDSLIGGNASAEGPEGE
GTESTVITGVDIVMNHHLQETSFTKEAYKKYIKDYMKSIKGKLEEQRPERVKPFMTGAAE
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>gi|27477073|gb|NM_018725.2|IL17BR 2077bp mRNA Homo sapiens interleukin 17B receptor (IL17BR), transcript variant 1, mRNA.
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CTTGTAAGAAGAATGAGGAGACAGTAGAAGTGAACTTCACAACCACTCCCCTGGGAAACA
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ACCAGAAGAAACAAACGCGAGCTTCAGTGGTGATTCCAGTGACTGGGGATAGTGAAGGTG
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AGAAGGCAGCAGACAAAGTCGTCTTCCTTCTTTCCAATGACGTCAACAGTGTGTGCGATG
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CCTTTAACCTTTTCTGCAGTGATCTAAGAAGCCAGATTCATCTGCACAAATACGTGGTGG
TCTACTTTAGAGAGATTGATACAAAAGACGATTACAATGCTCTCAGTGTCTGCCCCAAGT
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TGTCAGCAGGAAAAAGATCACAAGCCTGCCACGATGGCTGCTGCTCCTTGTAGCCCACCC
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CAATAAAATTTTCAAATATTGCTAACTAATGTAGCATTAACTAACGATTGGAAACTACAT
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AACTCTAAGAACCCCTCACTGATTCACTCAATAGCATCTTAAGTGAAAAACCTTCTATTA
CATGCAAAAAATCATTGTTTTTAAGATAACAAAAGTAGGGAATAAACAAGCTGAACCCAC
TTTTAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
>gi|27477074|gb|NP_061195.2|IL17BR 502aa linear IL-17B receptor isoform 1 precursor; IL-17B receptor; interleukin 17 receptor homolog 1; interleukin 17 receptor homolog; cytokine receptor CRL4 [Homo sapiens].
MSLVLLSLAALCRSAVPREPTVQCGSETGPSPEWMLQHDLIPGDLRDLRVEPVTTSVATG
DYSILMNVSWVLRADASIRLLKATKICVTGKSNFQSYSCVRCNYTEAFQTQTRPSGGKWT
FSYIGFPVELNTVYFIGAHNIPNANMNEDGPSMSVNFTSPGCLDHIMKYKKKCVKAGSLW
DPNITACKKNEETVEVNFTTTPLGNRYMALIQHSTIIGFSQVFEPHQKKQTRASVVIPVT
GDSEGATVQLTPYFPTCGSDCIRHKGTVVLCPQTGVPFPLDNNKSKPGGWLPLLLLSLLV
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SEVILEKWQKKKIAEMGPVQWLATQKKAADKVVFLLSNDVNSVCDGTCGKSEGSPSENSQ
DLFPLAFNLFCSDLRSQIHLHKYVVVYFREIDTKDDYNALSVCPKYHLMKDATAFCAELL
HVKQQVSAGKRSQACHDGCCSL
>gi|14165275|gb|NM_032411.1|ECRG4 772bp mRNA Homo sapiens esophageal cancer related gene 4 protein (ECRG4), mRNA.
GGATAACCCGCGGCCGCGCCTGCCCGCTCGCACCCCTCTCCCGCGCCCGGTTCTCCCTCG
CAGCACCTCGAAGTGCGCCCCTCGCCCTCCTGCTCGCGCCCCGCCGCCATGGCTGCCTCC
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GTTCCAACTAAGACTAAAGTGGCCGTTGATGAGAATAAAGCCAAAGAATTCCTTGGCAGC
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TACGATGACTACTAACCATGACTTGCCACACGCTGTACAAGAAGCAAATAGCGATTCTCT
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>gi|14165276|gb|NP_115787.1|ECRG4 148aa linear esophageal cancer related gene 4 protein [Homo sapiens].
MAASPARPAVLALTGLALLLLLCWGPGGISGNKLKLMLQKREAPVPTKTKVAVDENKAKE
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RHYDEDSAIGPRSPYGFRHGASVNYDDY

>gi|24025684|gb|NM_003017.2|SFRS3 1403bp mRNA Homo sapiens splicing factor, arginine/serine-rich 3 (SFRS3), mRNA.
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GCGTGTGGATTTGAGCCGCCGCATTTTTTAACCCTAGATCTCGAAATGCATCGTGATTCC
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TTGGAACGGGCTTTTGGCTACTATGGACCACTCCGAAGTGTGTGGGTTGCTAGAAACCCA
CCCGGCTTTGCTTTTGTTGAATTTGAAGATCCCCGAGATGCAGCTGATGCAGTCCGAGAG
CTAGATGGAAGAACACTATGTGGCTGCCGTGTAAGAGTGGAACTGTCGAATGGTGAAAAA
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GTCCTGCCAGTTTAAGGGTACATTGTAGAGCCGAACTTTGAGTTACTGTGCAAGATTTTT
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CAAATTGTTAAAAAAAAAAAAAA
>gi|4506901|gb|NP_003008.1|SFRS3 164aa linear splicing factor, arginine/serine-rich 3; splicing factor, arginine//serine-rich, 20-kD [Homo sapiens].
MHRDSCPLDCKVYVGNLGNNGNKTELERAFGYYGPLRSVWVARNPPGFAFVEFEDPRDAA
DAVRELDGRTLCGCRVRVELSNGEKRSRNRGPPPSWGRRPRDDYRRRSPPPRRRSPRRRS
FSRSRSRSLSRDRRRERSLSRERNHKPSRSFSRSRSRSRSNERK
>gi|4759097|gb|NM_004593.1|SFRS10 1972bp mRNA Homo sapiens splicing factor, arginine/serine-rich 10 (transformer 2 homolog, Drosophila) (SFRS10), mRNA.
GAATTCGGCACGAGGGCGACCGGCGCGTCGTGCGGGGCTGCGGCGGAGCCTCCTTAAGGA
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TGGAAGTGCTCACGGATCGGGGAAATCTGCAAGGCATACCCCTGCAAGGTCTCGCTCCAA
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CAGAAGAAGCTCCCGAAGGCATTATACCCGGTCACGGTCTCGCTCCCGCTCCCATAGACG
ATCACGTAGCAGGTCTTACAGTCGAGATTATCGTAGACGGCACAGCCACAGCCATTCTCC
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ATTTGCCTTTGTATATTTTGAAAATGTAGATGATGCCAAGGAAGCTAAAGAACGTGCCAA
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CAGAGGAGGAGGTGGAGGAGGAGGAGGATGGAGAGCTGCCCAAGACAGGGATCAGATTTA
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TGTTACAAGGGGATAATGGTATTTCAACTAGTTATCAGCAAGTGACAATACATTCCACCA
CAAATACACTCTTGTTCTTCTAGCTTTTAGACTATATGAAAAAACCGGGTGCTTCAAAGT
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>gi|4759098|gb|NP_004584.1|SFRS10 288aa linear splicing factor, arginine/serine-rich 10 (transformer 2 homolog, Drosophila); splicing factor, arginine/serine-rich (transformer 2 Drosophila homolog) 10 [Homo sapiens].
MSDSGEQNYGERESRSASRSGSAHGSGKSARHTPARSRSKEDSRRSRSKSRSRSESRSRS
RRSSRRHYTRSRSRSRSHRRSRSRSYSRDYRRRHSHSHSPMSTRRRHVGNRANPDPNCCL
GVFGLSLYTTERDLREVFSKYGPIADVSIVYDQQSRRSRGFAFVYFENVDDAKEAKERAN
GMELDGRRIRVDFSITKRPHTPTPGIYMGRPTYGSSRRRDYYDRGYDRGYDDRDYYSRSY
RGGGGGGGGWRAAQDRDQIYRRRSPSPYYSRGGYRSRSRSRSYSPRRY
>gi|5803206|gb|NM_006758.1|U2AF1 904bp mRNA Homo sapiens U2(RNU2) small nuclear RNA auxillary factor 1 (U2AF1), mRNA.
GGAATTCCGTCGACGGCAGCGGCGGCGGCGGGTGGGAAATGGCGGAGTATCTGGCCTCCA
TCTTCGGCACCGAGAAAGACAAAGTCAACTGTTCATTTTATTTCAAAATTGGAGCATGTC
GTCATGGAGACAGGTGCTCTCGGTTGCACAATAAACCGACGTTTAGCCAGACCATTGCCC
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ACCGTGGTCGTGGCGGTGGCGGTGGCGGTGGTGGAGGTGGCGGCGGACGGGAGCGTGACA
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AAAAACAACAAAAAAAAAACATACAAAGATGGGTTTCTGAATAAAAATTTGTAGTGATAA

CAGT
>gi|5803207|gb|NP_006749.1|U2AF1 240aa linear U2 small nuclear RNA auxillary factor 1; U2 snRNP auxiliary factor small subunit; splicing factor U2AF 35kDa subunit [Homo sapiens].
MAEYLASIFGTEKDKVNCSFYFKIGACRHGDRCSRLHNKPTFSQTIALLNIYRNPQNSSQ
SADGLRCAVSDVEMQEHYDEFFEEVFTEMEEKYGEVEEMNVCDNLGDHLVGNVYVKFRRE
EDAEKAVIDLNNRWFNGQPIHAELSPVTDFREACCRQYEMGECTRGGFCNFMHLKPISRE
LRRELYGRRRKKHRSRSRSRERRSRSRDRGRGGGGGGGGGGGRERDRRRSRDRERSGRF
>gi|23308726|gb|NM_003242.3|TGFBR2 2090bp mRNA Homo sapiens transforming growth factor, beta receptor II (70/80kDa) (TGFBR2), mRNA.
GTTGGCGAGGAGTTTCCTGTTTCCCCCGCAGCGCTGAGTTGAAGTTGAGTGAGTCACTCG
CGCGCACGGAGCGACGACACCCCCGCGCGTGCACCCGCTCGGGACAGGAGCCGGACTCCT
GTGCAGCTTCCCTCGGCCGCCGGGGGCCTCCCCGCGCCTCGCCGGCCTCCAGGCCCCTCC
TGGCTGGCGAGCGGGCGCCACATCTGGCCCGCACATCTGCGCTGCCGGCCCGGCGCGGGG
TCCGGAGAGGGCGCGGCGCGGAGCGCAGCCAGGGGTCCGGGAAGGCGCCGTCCGTGCGCT
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TGTGGCCGCTGCACATCGTCCTGTGGACGCGTATCGCCAGCACGATCCCACCGCACGTTC
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CCACGTGTGCCAACAACATCAACCACAACACAGAGCTGCTGCCCATTGAGCTGGACACCC
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ACAACGTGTTGAGAGATCGAGGGCGACCAGAAATTCCCAGCTTCTGGCTCAACCACCAGG
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TCACAGCCCAGTGTGTGGCAGAACGCTTCAGTGAGCTGGAGCATCTGGACAGGCTCTCGG
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>gi|23308727|gb|NP_003233.3|TGFBR2 567aa linear transforming growth factor, beta receptor II (70/80kDa); transforming growth factor, beta receptor II (70-80kD) [Homo sapiens].
MGRGLLRGLWPLHIVLWTRIASTIPPHVQKSVNNDMIVTDNNGAVKFPQLCKFCDVRFST
CDNQKSCMSNCSITSICEKPQEVCVAVWRKNDENITLETVCHDPKLPYHDFILEDAASPK
CIMKEKKKPGETFFMCSCSSDECNDNIIFSEEYNTSNPDLLLVIFQVTGISLLPPLGVAI
SVIIIFYCYRVNRQQKLSSTWETGKTRKLMEFSEHCAIILEDDRSDISSTCANNINHNTE
LLPIELDTLVGKGRFAEVYKAKLKQNTSEQFETVAVKIFPYEEYASWKTEKDIFSDINLK
HENILQFLTAEERKTELGKQYWLITAFHAKGNLQEYLTRHVISWEDLRKLGSSLARGIAH
LHSDHTPCGRPKMPIVHRDLKSSNILVKNDLTCCLCDFGLSLRLDPTLSVDDLANSGQVG
TARYMAPEVLESRMNLENAESFKQTDVYSMALVLWEMTSRCNAVGEVKDYEPPFGSKVRE
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LEHLDRLSGRSCSEEKIPEDGSLNTTK
>gi|5174728|gb|NM_006022.1|TSC22 1725bp mRNA Homo sapiens transforming growth factor beta-stimulated protein TSC-22 (TSC22), mRNA.
CGCCTCTTCACGGCACTGGGATCCGCATCTGCCTGGGATCATCAAGCCCTAGAAGCTGGG
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>gi|5174729|gb|NP_006013.1|TSC22 144aa linear transforming growth factor beta-stimulated protein TSC-22 [Homo sapiens].

MKSQWCRPVAMDLGVYQLRHFSISFLSSLLGTENASVRLDNSSSGASVVAIDNKIEQAMD
LVKSHLMYAVREEVEVLKEQIKELIEKNSQLEQENNLLKTLASPEQLAQFQAQLQTGSPP
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>gi|24432096|gb|NM_152912.2|MTIF3 1693bp mRNA Homo sapiens mitochondrial translational initiation factor 3 (MTIF3), mRNA.
GCAGATCCGCTGTACTTGCGGGCGCTACAGTATGTCAATCGCTTGCCCCAGCACAGTGGG
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GAATCAATACTCC
>gi|23097266|gb|NP_690876.1|MTIF3 278aa linear mitochondrial translational initiation factor 3 [Homo sapiens].
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TQNEGKKTKKNKTAFSNVGRKISQRVIHLFDEKGNDLGNMHRANVIRLMDERDLRLVQRN
TSTEPAEYQLMTGLQILQERQRLREMEKANPKTGPTLRKELILSSNIGQHDLDTKTKQIQ
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RALSKNEEKAYKETQETQERDTLNKDHGNDKESNVLHQ
>gi|27499034|gb|XM_044349.7|CAMK2G 1776bp mRNA Homo sapiens calcium/calmodulin-dependent protein kinase (CaM kinase) II gamma (CAMK2G), mRNA.
CAGCATGGCCACCACCGCCACCTGCACCCGTTTCACCGACGACTACCAGCTCTTCGAGGA
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CGGGCAGGGTCGGCCTCGCACCAGCCAGTCAGAAGAGACCCGGGTCTGGCACCGTCGGGA
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>gi|27499035|gb|XP_044349.7|CAMK2G 518aa linear similar to calcium/calmodulin -dependent protein kinase II gamma [Mus musculus] [Homo sapiens].

MATTATCTRFTDDYQLFEELGKGAFSVVRRCVKKTSTQEYAAKIINTKKLSARDHQKLER
EARICRLLKHPNIVRLHDSISEEGFHYLVFDLVTGGELFEDIVAREYYSEADASHCIHQI
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>gi|5453881|gb|NM_006213.1|PHKG1 1377bp mRNA Homo sapiens phosphorylase kinase, gamma 1 (muscle) (PHKG1), mRNA.

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>gi|5453882|gb|NP_006204.1|PHKG1 387aa linear phosphorylase kinase, gamma 1 (muscle) [Homo sapiens].
MTRDEALPDSHSAQDFYENYEPKEILGRGVSSVVRRCIHKPTSQEYAVKVIDVTGGGSFS
PEEVRELREATLKEVDILRKVSGHPNIIQLKDTYETNTFFFLVFDLMKRGELFDYLTEKV
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LREVCGTPSYLAPEIIECSMNEDHPGYGKEVDMWSTGVIMYTLLAGSPPFWHRKQMLMLR
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>gi|4503412|gb|NM_001945.1|DTR 2360bp mRNA Homo sapiens diphtheria toxin receptor (heparin-binding epidermal growth factor-like growth factor) (DTR), mRNA.
GCTACGCGGGCCACGCTGCTGGCTGGCCTGACCTAGGCGCGCGGGGTCGGGCGGCCGCGC
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>gi|4503413|gb|NP_001936.1|DTR 208aa linear diphtheria toxin receptor (heparin-binding epidermal growth factor-like growth factor); Diphtheria toxin receptor (heparin-binding EGF-like growth factor) [Homo sapiens].

MKLLPSVVLKLFLAAVLSALVTGESLERLRRGLAAGTSNPDPPTVSTDQLLPLGGGRDRK
VRDLQEADLDLLRVTLSSKPQALATPNKEEHGKRKKKGKGLGKKRDPCLRKYKDFCIHGE
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>gi|4507460|gb|NM_003236.1|TGFA 4119bp mRNA Homo sapiens transforming growth factor, alpha (TGFA), mRNA.

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>gi|4507461|gb|NP_003227.1|TGFA 160aa linear transforming
growth factor, alpha [Homo sapiens].
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splicing factor 3b, subunit 1, 155kDa (SF3B1), mRNA.
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>gi|6912654|gb|NP_036565.1|SF3B1 1304aa linear splicing factor 3b, subunit 1, 155kDa; spliceosome-associated factor 155; splicing factor 3b, subunit 1, 155kD [Homo sapiens].
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LKVVNGAAASQPPSKRKRRWDQTADQTPGATPKKLSSWDQAETPGHTPSLRWDETPGRAK
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GTPAMNMATPTPGHIMSMTPEQLQAWRWEREIDERNRPLSDEELDAMFPEGYKVLPPPAG
YVPIRTPARKLTATPTPLGGMTGFHMQTEDRTMKSVNDQPSGNLPFLKPDDIQYFDKLLV
DVDESTLSPEEQKERKIMKLLLKIKNGTPPMRKAALRQITDKAREFGAGPLFNQILPLLM
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AKAAGLATMISTMRPDIDNMDEYVRNTTARAFAVVASALGIPSLLPFLKAVCKSKKSWQA
RHTGIKIVQQIAILMGCAILPHLRSLVEIIEHGLVDEQQKVRTISALAIAALAEAATPYG
IESFDSVLKPLWKGIRQHRGKGLAAFLKAIGYLIPLMDAEYANYYTREVMLILIREFQSP
DEEMKKIVLKVVKQCCGTDGVEANYIKTEILPPFFKHFWQHRMALDRRNYRQLVDTTVEL
ANKVGAAEIISRIVDDLKDEAEQYRKMVMETIEKIMGNLGAADIDHKLEEQLIDGILYAF
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VMKTCQEEKLMGHLGVVLYEYLGEEYPEVLGSILGALKAIVNVIGMHKMTPPIKDLLPRL
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FGYIAKAIGPHDVLATLLNNLKVQERQNRVCTTVAIAIVAETCSPFTVLPALMNEYRVPE
LNVQNGVLKSLSFLFEYIGEMGKDYIYAVTPLLEDALMDRDLVHRQTASAVVQHMSLGVY
GFGCEDSLNHLLNYVWPNVFETSPHVIQAVMGALEGLRVAIGPCRMLQYCLQGLFHPARK
VRDVYWKIYNSIYIGSQDALIAHYPRIYNDDKNTYIRYELDYIL
>gi|21707321|gb|BC033864.1|BC033864 2321bp mRNA Homo sapiens, Similar to branched chain aminotransferase 1, cytosolic, clone MGC:45234 IMAGE:5186262, mRNA, complete cds.
GGTGGATGCTGCGGCATCGGAGGACCCTGCTGGTGGAGGAAATGGTTCACGCCCGTCCCC
GTTCCCTTTGCAGGCTTGCTATTGTGCGTCTGTGATTGACAAGACCACGAGGCTGAGCGC
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ACTTTGGCACTTTGGGAGGCTGAGGTGGGAGGATCACATGAGCCCAGCCTGGGCAACATA
GGGAGACCGCTATCTTTACAAAATAAAATATAAAATATAAAACCTAGTTGGGCATGGCAG
CATACACCTGTAGTCCCAGGTGCTCGGGAGACTGAGACAGGAGGATCGCTTGGGCCTGGG
AGGTCGAGGCTGCAGTGAGCCAAGATTATGCCACTGCATTCCAGCCTGGGTGACAGGGCA
AGACCCTGTCTTAAAAAAAAAAAAAAAAAAAAAAAAAAAA
>gi|21707322|gb|AAH33864.1|AAH33864 320aa linear Similar to branched chain aminotransferase 1, cytosolic [Homo sapiens].
MDCSNGCSAECTGEGGSKEVVGTFKAKDLIVTPATILKEKPDPNNLVFGTVFTDHMLTVE
WSSEFGWEKPHIKPLQNLSLHPGSSALHYAVELFEGLKAFRGVDNKIRLFQPNLNMDRMY
RSAVRATLPVFDKEELLECIQQLVKLDQEWVPYSTSASLYIRPTFIGTEPSLGVKKPTKA
LLFVLLSPVGPYFSSGTFNPVSLWANPKYVRAWKGGTGDCKMGGNYGSSLFAQCEAVDNG
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DTELSLFSINLPDFLQFIYF
>gi|29570794|gb|NM_001895.2|CSNK2A1 2323bp mRNA Homo sapiens casein kinase 2, alpha 1 polypeptide (CSNK2A1), transcript variant 2, mRNA.
CCCGCCTCCTGGTAGGAGGGGGTTTCCGCTTCCGGCAGCAGCGGCTGCAGCCTCGCTCTG
GTCCCTGCGGCTGGCGGCCGAGCCGTGTGTCTCCTCCTCCATCGCCGCCATATTGTCTGT
GTGAGCAGAGGGGAGAGCGGCCGCCGCCGCTGCCGCTTCCACCACAGTTTGAAGAAAACA
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TATCTCGTGCTCTTCTCCAGAGATTACAAAAATGTAGCTTCTCAGGGGAGGCAGGAAGAA
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>gi|4503095|gb|NP_001886.1|CSNK2A1 391aa linear casein kinase II alpha 1 subunit isoform a; CK2 catalytic subunit alpha [Homo sapiens].

MSGPVPSRARVYTDVNTHRPREYWDYESHVVEWGNQDDYQLVRKLGRGKYSEVFEAINIT
NNEKVVVKILKPVKKKKIKREIKILENLRGGPNIITLADIVKDPVSRTPALVFEHVNNTD
FKQLYQTLTDYDIRFYMYEILKALDYCHSMGIMHRDVKPHNVMIDHEHRKLRLIDWGLAE
FYHPGQEYNVRVASRYFKGPELLVDYQMYDYSLDMWSLGCMLASMIFRKEPFFHGHDNYD
QLVRIAKVLGTEDLYDYIDKYNIELDPRFNDILGRHSRKRWERFVHSENQHLVSPEALDF
LDKLLRYDHQSRLTAREAMEHPYFYTVVKDQARMGSSSMPGGSTPVSSANMMSGISSVPT
PSPLGPLAGSPVIAAANPLGMPVPAAAGAQQ

>gi|13375963|gb|NM_024689.1|FLJ14103 2502bp mRNA Homo sapiens hypothetical protein FLJ14103 (FLJ14103); mRNA.

CTCTTTGGCCAAGCCCTGCCTCTGTACAGCCTCGAGTGGACAGCCAGAGGCTGCAGCTGG
AGCCCAGAGCCCAAGATGGAGCCCCAGCTGGGGCCTGAGGCTGCCGCCCTCCGCCCTGGC
TGGCTGGCCCTGCTGCTGTGGGTCTCAGCCCTGAGCTGTTCTTTCTCCTTGCCAGCTTCT
TCCCTTTCTTCTCTGGTGCCCCAAGTCAGAACCAGCTACAATTTTGGAAGGACTTTCCTC
GGTCTTGATAAATGCAATGCCTGCATCGGGACATCTATTTGCAAGAAGTTCTTTAAAGAA
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CCAAAGACCTGCAGCATTGAGCGTGTCCTGCGGAAAACAGAGAGGTTCCAGAAATGGCTG
CAGGCCAAGCGCCTCACGCCGGACCTGGTGCAGGACTGTCACCAGGGCCAGAGAGAACTA
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GACAACTCCAAAGCCCCGGCTCTTTCCACCACACTGTGGTCCCCTAGATGGGGCTGTTGC
TGAGCCCACCCCAATCCAGATGTGATCCCCCTGTGATCTACTTCTGGCAAGATTCTCAGT
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TAATTGTGGGTTTGCACATGGCCAGCACATGCCAAACAGAAGTAGACAAAGGTCTCACTC
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TAGCAGTTATCTATTGTTGTGTATTAAACCATTTCAACACAT

>gi|13375964|gb|NP_078965.1|FLJ14103 182aa linear hypothetical protein FLJ14103 [Homo sapiens].

MEPQLGPEAAALRPGWLALLLWVSALSCSFSLPASSLSSLVPQVRTSYNFGRTFLGLDKC
NACIGTSICKKFFKEEIRSDNWLASHLGLPPDSLLSYPANYSDDSKIWRPVEIFRLVSKY
QNEISDRKICASASAPKTCSIERVLRKTERFQKWLQAKRLTPDLVQDCHQGQRELKFLCM
LR

>gi|7658290|gb|AF221842.1|AF221842 3057bp mRNA Homo sapiens U5 snRNP-associated 102 kDa protein mRNA, complete cds.

ACTTTGCTACGGAGTGCATCGGACGTCGAAGCCTAGAGTCTCTGCGTCTTTCCCTCTTCC
GCTGCCTCATTCCTTTCCTTCCTAGCCTTGGTCGTCGCCGCCACCATGAACAAGAAGAAG
AAACCGTTCCTAGGGATGCCCGCGCCCCTCGGCTACGTGCCGGGGCTGGGCCGGGGCGCC
ACTGGCTTCACCACGCGGTCAGACATTGGGCCCGCCCGTGATGCAAATGACCCTGTGGAT
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GCTGACGATGACGACGAGGATCTAAATGACACCAATTACGATGAGTTTAATGGCTATGCT
GGGAGCCTCTTCTCAAGTGGACCCTACGAGAAAGATGATGAGGAAGCAGATGCTATCTAT
GCAGCCCTGGATAAAAGGATGGATGAAAGAAGAAAAGAAAGACGGGAGCAAAGGGAGAAA
GAAGAAATAGAGAAATATCGTATGGAACGCCCCAAAATCCAACAGCAGTTCTCAGACCTC
AAGAGGAAGTTGGCAGAAGTCACAGAAGAAGAGTGGCTGAGCATCCCCGAGGTTGGCGAT
GCCAGAAATAAACGTCAGCGGAACCCACGCTATGAGAAGCTGACCCCTGTTCCTGACAGT
TTCTTTGCCAAACATTTACAGACCGGAGAGAACCATACCTCAGTGGATCCCCGACAAACT
CAATTTGGAGGTCTTAACACACCCTATCCAGGTGGACTAAACACTCCATACCCAGGTGGA
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GCGAGGAACACTCTGATGGACATGAGGCTGAGCCAGGTGTCTGACTCCGTGAGTGGACAG
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CAAGTAGCTCGGAACCTTATCATGAAGGGGACGGAGATGTGCCCCAAGAGTGAAGATGTC
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>gi|7658291|gb|AAF66128.1|AAF66128 941aa linear U5 snRNP-associated 102 kDa protein [Homo sapiens].

MNKKKKPFLGMPAPLGYVPGLGRGATGFTTRSDIGPARDANDPVDDRHAPPGKRTVGDQM
KKNQAADDDDEDLNDTNYDEFNGYAGSLFSSGPYEKDDEEADAIYAALDKRMDERRKERR
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PVPDSFFAKHLQTGENHTSVDPRQTQFGGLNTPYPGGLNTPYPGGMTPGLMTPGTGELDM
RKIGQARNTLMDMRLSQVSDSVSGQTVVDPKGYLTDLNSMIPTHGGDINDIKKARLLLKS
VRETNPHHPPAWIASARLEEVTGKLQVARNLIMKGTEMCPKSEDVWLEAARLQPGDTAKA
VVAQAVRHLPQSVRIYIRAAELETDIRAKKRVLRKALEHVPNSVRLWKAAVELEEPEDAR
IMLSRAVECCPTSVELWLALARLETYENARKVLNKARENIPTDRHIWITAAKLEEANGNT
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VAHCPKAEVLWLMGAKSKWLAGDVPAARSILALAFQANPNSEEIWLAAVKLESENDEYER
ARRLLAKARSSAPTARVFMKSVKLEWVQDNIRAAQDLCEEALRHYEDFPKLWMMKGQIEE

QKEMMEKAREAYNQGLKKCPHSTPLWLLLSRLEEKIGQLTRARAILEKSRLKNPKNPGLW
LESVRLEYRAGLKNIANTLMAKALQECPNSGILWSEAIFLEARPQRRTKSVDALKKCEHD
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CESAEPRHGELWCAVSKDIANWQKKIGDILRLVAGRIKNTF
>gi|5454165|gb|NM_006370.1|VTI1B 1287bp mRNA Homo sapiens vesicle transport through interaction with t-SNAREs homolog 1B (yeast) (VTI1B), mRNA.
CCCTTTCGCTGCGGCCTTTCCCCAACCCGGACCCGGCACTTCTCGGGTTCCGCGACTGCC
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>gi|5454166|gb|NP_006361.1|VTI1B 232aa linear vesicle-associated soluble NSF attachment protein receptor (v-SN; vesicle-associated soluble NSF attachment protein receptor (v-SNARE; homolog of S. cerevisiae VTI1) [Homo sapiens].
MASSAASSEHFEKLHEIFRGLHEDLQGVPERLLGTAGTEEKKKLIRDFDEKQQEANETLA
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LVNTSENLSKSRKILRSMSRKVTTNKLLLSIIILLELAILGGLVYYKFFRSH
>gi|7705992|gb|NM_016440.1|LOC51231 1869bp mRNA Homo sapiens VRK3 for vaccinia related kinase 3 (LOC51231), mRNA.
CCGAGGGTCAGGCTGCAGAAGCCCAGAATCCCACCCCAGTCCCCAAGTACAGAGGTCGCT
GTCAAGATGGAGTTTCCAACCCAGTAAATCCAAGGGCCAGACCGTGACCTCATAAAGCAT
GATCTCCTTCTGTCCAGACTGTGGCAAAAGTATCCAAGCGGCATTCAAATTCTGCCCCTA
CTGTGGAAATTCTTTGCCTGTAGAGGAGCATGTAGGGTCCCAGACCTTTGTCAATCCACA
TGTGTCATCCTTCCAAGGCTCAAAGAGAGGGCTGAACTCCAGTTTTGAAACCTCTCCTAA
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CAGTTCTGAGTCTGAAGATACTCTGAGTTCCTCTGAGAGATCCAAAGGCTCCGGGAGCAG
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TAGCCCTCAGAAGACCAGCTGTAGCCCTCAGAAGACCAGGCAGAGCCCTCAGACGCTGAA
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GAGTGGGCGACAGTGGAAGCTGAAGTCCTTCCAGACCAGGGACAACCAGGGCATTCTCTA
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CCTGGGGAGGAGCCTTCAGTCGGCCCTGGATGTCAGCCCAAAGCATGTGCTGTCAGAGAG
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GTATGTTCATGGAAATGTGACAGCTGAAAATATCTTTGTGGATCCAGAGGACCAGAGTCA
GGTGACTTTGGCAGGCTATGGCTTCGCCTTCCGCTATTGCCCAAGTGGCAAACACGTGGC
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GCACAAGGGATGCGGGCCCTCCCGCCGCAGCGACCTCCAGAGCCTGGGCTACTGCATGCT
GAAGTGGCTCTACGGGTTTCTGCCATGGACAAATTGCCTTCCCAACACTGAGGACATCAT
GAAGCAAAAACAGAAGTTTGTTGATAAGCCGGGGCCCTTCGTGGGACCCTGCGGTCACTG
GATCAGGCCCTCAGAGACCCTGCAGAAGTACCTGAAGGTGGTGATGGCCCTCACGTATGA
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TGTGTCTCCATATGACCCCATTGGCCTCCCGATGGTGCCCTAGGTGGAATCCAGAACTTT
CCATTTGCAGTGTGCAACAGAAAAAAAAATGAAGCAATGTGACTCAAGGCCTGCTGTTTA
ATCACAGATAAGCTTCTAGAACAAGCCCTGGAATGTGCATTCCTGCCACTGGTTTCAGGA
TACTCATCAGTCCTGATTAGCCTCCGGAGGGCCCCAGTTTCCCTCCCGTGAATGTGAAGT
TCCCCATCTTGGTGGCCTGCCCTTCAGCCAGTGTCCTAGCAAAGCTGGATGGGGTTGGGC
CGGCCCACAGGGGGGACCCCTCCTACCCTTGACTCCTCTGTGCTTTGGTAATAAATTGTT
TTACCAGAG

>gi|7705993|gb|NP_057524.1|LOC51231 474aa linear VRK3 for vaccinia related kinase 3 [Homo sapiens].

MISFCPDCGKSIQAAFKFCPYCGNSLPVEEHVGSQTFVNPHVSSFQGSKRGLNSSFETSP
KKVKWSSTVTSPRLSLFSDGDSSESEDTLSSSERSKGSGSRPPTPKSSPQKTRKSPQVTR
GSPQKTSCSPQKTRQSPQTLKRSRVTTSLEALPTGTVLTDKSGRQWKLKSFQTRDNQGIL
YEAAPTSTLTCDSGPQKQKFSLKLDAKDGRLFNEQNFFQRAAKPLQVNKWKKLYSTPLLA
IPTCMGFGVHQDKYRFLVLPSLGRSLQSALDVSPKHVLSERSVLQVACRLLDALEFLHEN
EYVHGNVTAENIFVDPEDQSQVTLAGYGFAFRYCPSGKHVAYVEGSRSPHEGDLEFISMD
LHKGCGPSRRSDLQSLGYCMLKWLYGFLPWTNCLPNTEDIMKQKQKFVDKPGPFVGPCGH
WIRPSETLQKYLKVVMALTYEEKPPYAMLRNNLEALLQDLRVSPYDPIGLPMVP

>gi|27479296|gb|XM_114075.2|TCEA3 1543bp mRNA Homo sapiens transcription elongation factor A (SII), 3 (TCEA3), mRNA.

CGCCCCCGCCGGGCGTGTGTGTCGTGTGTGTTTGGGGCCCGCGCGGGTTGCGCGCCCTCC
GCCTTCGCGCCTCCTGCCCCCGAGGCCCTACTGCTGCCCCTGTGCCCCTCGCCCCGCCGG
GCGTCGCGGGCCAACATGGGCCAGGAAGAGGAGCTGCTGAGGATCGCCAAAAAGCTGGAG
AAGATGGTGGCCAGGAAGAACACGGAAGGGGCCCTGGACCTTCTGAAGAAGCTGCACAGC
TGCCAGATGTCCATCCAGCTACTACAGACAACCAGGATTGGAGTTGCTGTTAATGGGGTC
CGCAAGCACTGCTCAGACAAGGAGGTGGTGTCCTTGGCCAAAGTCCTTATCAAAAACTGG
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GCATCAGAAATCGAAGATCATATCTACCAAGAGCTCAAGAGCACGGACATGAAGTACCGG
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ACAAGGTGAGGAAGAAGAAAGAGGAAGCGCTGAATTATCTGAACTGGAGAAGCAATAAAA
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ATTCTCTTGCAAATTAATAATCGGTCATTAGAAACAATTGGTTAATGGGGGAGCCTAATT
GGAGAATGATGCTGAGAATTTGTATTGATGAACCTCTTTTAGAAACTGCAGAGGGCTGGG
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ACCCCAGAAGTTTGAGTCCAGCCCAGGCAACACAGCAAGACCC

>gi|20473950|gb|XP_114075.1|TCEA3 348aa linear similar to Transcription elongation factor A protein 3 (Transcription elongation factor S-II protein 3) (Transcription elongation factor TFIIS.h) [Homo sapiens].

MGQEEELLRIAKKLEKMVARKNTEGALDLLKKLHSCQMSIQLLQTTRIGVAVNGVRKHCS
DKEVVSLAKVLIKNWKRLLDSPGPPKGEKGEEREKAKKKEKGLECSDWKPEAGLSPPRKK
REDPKTRRDSVDSKSSASSSPKRPSVERSNSSKSKAESPKTPSSPLTPTFASSMCLLAPC
YLTGDSVRDKCVEMLSAALKADDDYKDYGVNCDKMASEIEDHIYQELKSTDMKYRNRVRS
RISNLKDPRNPGLRRNVLSGAISAGLIAKMTAEEMASDELRELRNAMTQEAIREHQMAKT
GGTTTDLFQCSKCKKKNCTYNQVQTRSADEPMTTFVLCNECGNRWKFC

>gi|21314607|gb|NM_003342.2|UBE2G1 2430bp mRNA Homo sapiens ubiquitin-conjugating enzyme E2G 1 (UBC7 homolog, C. elegans) (UBE2G1), mRNA.

ACCGGCAGCGAGGCGCCGCTCCCGCCGCCTCAGCCCGGCCTTCCTCGGCTCCGGCGCTCC
GGTCGCGGGGCCCGGGTTCCTCGGCACACCCCGCTCCAGCCGCCCCCAGAGCCTGTCCCC
AGCCCTTCGGAAGCCCCGGCGCCAGCCCGGGCCCTCGGCAGGGAGGATGACGGAGCTGCA
GTCGGCACTGCTACTGCGAAGACAGCTGGCAGAACTCAACAAAAATCCAGTGGAAGGCTT
TTCTGCAGGTTTAATAGATGACAATGATCTCTACCGATGGGAAGTCCTTATTATTGGCCC
TCCAGATACACTTTATGAAGGTGGTGTTTTTAAGGCTCATCTTACTTTCCCAAAAGATTA
TCCCCTCCGACCTCCTAAAATGAAATTCATTACAGAAATCTGGCACCCAAATGTTGATAA
AAATGGTGATGTGTGCATTTCTATTCTTCATGAGCCTGGGGAAGATAAGTATGGTTATGA
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CCAATTGAGAAACATGGCACTGTTTTTCCTGCACTCTACCCACCTATTGCTGGACTTCTG
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TGCTGACAAGAGCCTAACAAGTGCCAACTTACAGATGATTACGCATTTTGAATTCTAATG
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CAGAATGTTCTCAAATGATTTAGTTCAAATACTGAAATACTTAATGAGCAATTACTTGAT
TTTTAATGATGACTTCGAAGGAGTCATCACTAGGTGCTTTGTCCTTTTTGTATTCTAGTT
GCACCCACCTCTTGGATTGGATATAGCAATAACATTTATTGGCCGTTGTGAGCTCTTGAT
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CATGTTTGTTGTGAGGTTGTGTGTGGGAAGTAGTCTTTGGCTTATAAGGGATAGAACTTG
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CTGCTCTGTGGGGTTTGGAGAAATGCTTTGGCAGAAGAGTGAAAGAACTCCTGCCAAGAG
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AAAAAAAAAAAAAAAAAAAAAAAAAAAAA

>gi|13489085|gb|NP_003333.1|UBE2G1 170aa linear ubiquitin-conjugating enzyme E2G 1 (UBC7 homolog, C. elegans); Ubiquitin-conjugating enzyme E2G (UBC7, C. elegans, human homolog of); ubiquitin-conjugating enzyme E2G (homologous to C. elegans UBC7); ubiquitin-conjugating enzyme E2G 1 (homologous to C. elegans UBC7) [Homo sapiens].

MTELQSALLLRRQLAELNKNPVEGFSAGLIDDNDLYRWEVLIIGPPDTLYEGGVFKAHLT
FPKDYPLRPPKMKFITEIWHPNVDKNGDVCISILHEPGEDKYGYEKPEERWLPIHTVETI
MISVISMLADPNGDSPANVDAAKEWREDRNGEFKRKVARCVRKSQETAFE

>gi|21361498|gb|NM_015670.2|SENP3 2258bp mRNA Homo sapiens sentrin/SUMO-specific protease 3 (SENP3), mRNA.

GAAGCTTGAGGCCGGAGACGCCCGCCTTCGGGCCCGTCCGCCCGGCTTCCCCGCTCCCGG
GTACTGGAAGATGAAAGAGACTATACAAGGGACCGGGTCCTGGGGGCCTGAGCCTCCTGG
ACCCGGCATACCCCCAGCTTACTCAAGTCCCAGGCGGGAGCGTCTTCGTTGGCCCCCACC
TCCCAAACCCCGACTCAAGTCAGGTGGAGGGTTTGGGCCAGATCCTGGGTCAGGGACCAC
AGTGCCAGCCAGACGCCTCCCTGTCCCCCGACCCTCTTTTGATGCCTCAGCAAGTGAAGA
GGAGGAAGAAGAGGAGGAGGAGGAGGATGAAGATGAAGAGGAGGAAGTGGCAGCTTGGAG
GCTGCCCCCAAGATGGAGTCAGCTGGGAACCTCCCAGCGGCCCCGCCCTTCCCGCCCCAC
TCATCGAAAAACCTGCTCACAGCGCCGCCGCCGAGCCATGAGAGCCTTCCGGATGCTGCT
CTACTCAAAAAGCACCTCGCTGACATTCCACTGGAAGCTTTGGGGGCGCCACCGGGGCCG
GCGGCGGGGCCTCGCACACCCCAAGAACCATCTTTCACCCCAGCAAGGGGGTGCGACGCC
ACAGGTGCCATCCCCCTGTTGTCGTTTTGACTCCCCCCGGGGGCCACCTCCACCCCGGCT
GGGTCTGCTAGGTGCTCTCATGGCTGAGGATGGGGTGAGAGGGTCTCCACCAGTGCCCTC
TGGGCCCCCCATGGAGGAAGATGGACTCAGGTGGACTCCAAAGTCTCCTCTGGACCCTGA
CTCGGGCCTCCTTTCATGTACTCTGCCCAACGGTTTTGGGGGACAATCTGGGCCAGAAGG

GGAGCGCAGCTTGGCACCCCCTGATGCCAGCATCCTCATCAGCAATGTGTGCAGCATCGG
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ACAGAGCATCTTGGACGAATTCCTTCAAACGTATGGCAGCCTCATACCCCTCAGCACTGA
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CAAACTTCGTCGGCAGATCTACAAGGAGCTGTGTCACTGCAAACTCACTGTGTGAGCCTC
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CTCCAGTTCCTTTCCTCTCTTGCCTCTTCCCACTCACTTCCCTTTGGTTTTTCATATTTA
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GGGTGGTGGGCGGACAATAGGATCACTGCCTGCCAGATCTTCAAACTTTTATATATATAT
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AAGGATCCTTTGTTGATACGTAAAAAAAAAAAAAAAAA

>gi|21361499|gb|NP_056485.2|SENP3 574aa linear sentrin/SUMO-specific protease 3 [Homo sapiens].

MKETIQGTGSWGPEPPGPGIPPAYSSPRRERLRWPPPPKPRLKSGGGFGPDPGSGTTVPA
RRLPVPRPSFDASASEEEEEEEEEDEDEEEEVAAWRLPPRWSQLGTSQRPRPSRPTHRK
TCSQRRRRAMRAFRMLLYSKSTSLTFHWKLWGRHRGRRRGLAHPKNHLSPQQGGATPQVP
SPCCRFDSPRGPPPPRLGLLGALMAEDGVRGSPPVPSGPPMEEDGLRWTPKSPLDPDSGL
LSCTLPNGFGGQSGPEGERSLAPPDASILISNVCSIGDHVAQELFQGSDLGMAEEAERPG
EKAGQHSPLREEHVTCVQSILDEFLQTYGSLIPLSTDEVVEKLEDIFQQEFSTPSRKGLV
LQLIQSYQRMPGNAMVRGFRVAYKRHVLTMDDLGTLYGQNWLNDQVMNMYGDLVMDTVPE
KVHFFNSFFYDKLRTKGYDGVKRWTKNVDIFNKELLLIPIHLEVHWSLISVDVRRRTITY
FDSQRTLNRRCPKHIAKYLQAEAVKKDRLDFHQGWKGYFKMNVARQNNDSDCGAFVLQYC
KHLALSQPFSFTQQDMPKLRRQIYKELCHCKLTV

>gi|5803166|gb|NM_006802.1|SF3A3 2733bp mRNA Homo sapiens splicing factor 3a, subunit 3, 60kDa (SF3A3), mRNA.

AAGGGAAGATGGAGACAATACTGGAGCAGCAGCGGCGCTATCATGAGGAGAAGGAACGGC
TCATGGACGTCATGGCTAAAGAGATGCTCACCAAGAAGTCCACGCTCCGGGACCAGATCA
ATTCTGATCACCGCACTCGGGCCATGCAAGATAGGTATATGGAGGTCAGTGGGAACCTGA
GGGATTTGTATGATGATAAGGATGGATTACGAAAGGAGGAGCTCAATGCCATTTCAGGAC
CCAATGAGTTTGCTGAATTCTATAATAGACTCAAGCAAATAAAGGAATTCCACCGGAAGC
ACCCAAATGAGATCTGTGTGCCAATGTCAGTGGAATTTGAGGAACTCCTGAAGGCTCGAG
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GTTATCTCGATCTCCATGACTGTTACCTCAAGTACATTAACCTGAAGGCATCTGAGAAGC
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AGAATGCAGAGTATAAGAGATACCTAGAGATGCTGCTTGAGTACCTTCAGGATTACACAG
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CCCATGCTGGAGCCCATCTTGACCTCTCTGCATTCTCCTCCTGGGAGGAGTTGGCTTCTC
TGGGTTTGGACAGATTGAAATCTGCTCTCTTAGCTTTAGGCTTGAAATGTGGCGGGACCC
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CCTCTTTGTTTGCCAAAAATCCCAAGTCAAAGGGCACCAAGCGAGACACTGAAAGGAACA
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ACCCTGTCTCATTAAAAAAAAAAAAAAAAGGCATGGTGGCACGCACTGTAGTCCCAGCTA
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>gi|5803167|gb|NP_006793.1|SF3A3 501aa linear splicing factor 3a, subunit 3, 60kDa; pre-mRNA splicing factor SF3a (60kD) [Homo sapiens].

METILEQQRRYHEEKERLMDVMAKEMLTKKSTLRDQINSDHRTRAMQDRYMEVSGNLRDL
YDDKDGLRKEELNAISGPNEFAEFYNRLKQIKEFHRKHPNEICVPMSVEFEELLKARENP
SEEAQNLVEFTDEEGYGRYLDLHDCYLKYINLKASEKLDYITYLSIFDQLFDIPKERKNA
EYKRYLEMLLEYLQDYTDRVKPLQDQNELFGKIQAEFEKKWENGTFPGWPKETSSALTHA
GAHLDLSAFSSWEELASLGLDRLKSALLALGLKCGGTLEERAQRLFSTKGKSLESLDTSL
FAKNPKSKGTKRDTERNKDIAFLEAQIYEYVEILGEQRHLTHENVQRKQARTGEEREEEE
EEQISESESEDEENEIIYNPKNLPLGWDGKPIPYWLYKLHGLNINYNCEICGNYTYRGPK

AFQRHFAEWRHAHGMRCLGIPNTAHFANVTQIEDAVSLWAKLKLQKASERWQPDTEEEYE
DSSGNVVNKKTYEDLKRQGLL
>gi|28882054|gb|NM_005011.2|NRF1 2514bp mRNA Homo sapiens
nuclear respiratory factor 1 (NRF1), mRNA.
GAGGCTGCGAGGAGCCGGCGCGGTCGCAGTCTCCACGGCGCAGGCCCACGGTAGCGCAGC
CGCTCTGAGTAGAACTTCATGGAGGAACACGGAGTGACCCAAACCGAACATATGGCTACC
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CATAGTATGCTGAGTGCTGATGAAGACTCGCCTTCTTCTCCCGAGGACACCTCTTACGAT
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GCCCCTGCGCCACAGGAGGTTAACTCAGAACTGCCGCCTCTCACCATCGACGGAATTCCA
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CATGGAATTATCTGTATGAAATCAAGGTGCGCTGTGGAAACAATAATTCACCCAGTTTAG
TGGGTGGTAGGGTACGTGGCCAGACACAGTCACCCAGTTTTTGTTCATACCAGGGTCATG
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>gi|28882055|gb|NP_005002.2|NRF1 522aa linear nuclear respiratory factor 1 [Homo sapiens].
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>gi|6996000|gb|NM_001663.2|ARF6 1806bp mRNA Homo sapiens ADP-ribosylation factor 6 (ARF6), mRNA.
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GATCCATGACCTGACGGGGCCCCGGAGCCGCGCTGCCTCTCGGGTGTCCTGGGTCGGTGG
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ATAAAC
>gi|4502211|gb|NP_001654.1|ARF6 175aa linear ADP-ribosylation factor 6 [Homo sapiens].
MGKVLSKIFGNKEMRILMLGLDAAGKTTILYKLKLGQSVTTIPTVGFNVETVTYKNVKFN

VWDVGGQDKIRPLWRHYYTGTQGLIFVVDCADRDRIDEARQELHRIINDREMRDAIILIF
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>gi|23510442|gb|NM_003809.2|TNFSF12 1407bp mRNA Homo sapiens tumor necrosis factor (ligand) superfamily, member 12 (TNFSF12), transcript variant 1, mRNA.
CTCTCCCCGGCCCGATCCGCCCGCCGGCTCCCCCTCCCCCGATCCCTCGGGTCCCGGGAT
GGGGGGGCGGTGAGGCAGGCACAGCCCCCCGCCCCCATGGCCGCCCGTCGGAGCCAGAGG
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GTCATAAAAAAAAAAAAAAAAAAAAA
>gi|4507597|gb|NP_003800.1|TNFSF12 249aa linear tumor necrosis factor (ligand) superfamily, member 12 isoform 1 precursor; APO3/DR3 ligand; TNF-related WEAK inducer of apoptosis [Homo sapiens].
MAARRSQRRRGRRGEPGTALLVPLALGLGLALACLGLLLAVVSLGSRASLSAQEPAQEEL
VAEEDQDPSELNPQTEESQDPAPFLNRLVRPRRSAPKGRKTRARRAIAAHYEVHPRPGQD
GAQAGVDGTVSGWEEARINSSSPLRYNRQIGEFIVTRAGLYYLYCQVHFDEGKAVYLKLD
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TYFGLFQVH
>gi|11496238|gb|NM_021975.1|RELA 2444bp mRNA Homo sapiens v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian) (RELA), mRNA.
GGCACGAGGCGGGGCCGGGTCGCAGCTGGGCCCGCGGCATGGACGAACTGTTCCCCCTCA
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>gi|11496239|gb|NP_068810.1|RELA 537aa linear v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65; v-rel avian reticuloendotheliosis viral oncogene homolog A (nuclear factor of kappa light polypeptide gene enhancer in B-cells 3 (p65)) [Homo sapiens].

MDELFPLIFPAEQPKQRGMRFRYKCEGRSAGSIPGERSTDTTKTHPTIKINGYTGPGTVR
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KKSPFSGPTDPRPPPRRIAVPSRSSASVPKPAPQPYPFTSSLSTINYDEFPTMVFPSGQI
SQASALAPAPPQVLPQAPAPAPAPAMVSALAQAPAPVPVLAPGPPQAVAPPAPKPTQAGE
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>gi|23312372|gb|NM_001065.2|TNFRSF1A 2236bp mRNA Homo sapiens tumor necrosis factor receptor superfamily, member 1A (TNFRSF1A), mRNA.
GCTGTTGCAACACTGCCTCACTCTTCCCCTCCCACCTTCTCTCCCCTCCTCTCTGCTTTA
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AAAAAAAAAAAAAAA
>gi|4507575|gb|NP_001056.1|TNFRSF1A 455aa linear tumor necrosis factor receptor 1 precursor; tumor necrosis factor

receptor type 1; tumor necrosis factor-alpha receptor; tumor necrosis factor binding protein 1 [Homo sapiens].
MGLSTVPDLLLPLVLLELLVGIYPSGVIGLVPHLGDREKRDSVCPQGKYIHPQNNSICCT
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>gi|4506738|gb|NM_003952.1|RPS6KB2 1735bp mRNA Homo sapiens ribosomal protein S6 kinase, 70kDa, polypeptide 2 (RPS6KB2), mRNA.
AGAGACTCGTGCCGAATGGCACGAGGCCGACGGGCCCGCGGGGCCGGCGCCGCCATGGCG
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>gi|4506739|gb|NP_003943.1|RPS6KB2 495aa linear ribosomal protein S6 kinase, 70kDa, polypeptide 2; ribosomal protein S6 kinase, 70kD, polypeptide 2; p70 ribosomal S6 kinase beta [Homo sapiens].
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VELTETSVNVGPERIGPHCFELLRVLGKGGYGKVFQVRKVQGTNLGKIYAMKVLRKAKIV
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>gi|11995473|gb|NM_019884.1|GSK3A 2169bp mRNA Homo sapiens
glycogen synthase kinase 3 alpha (GSK3A), mRNA.
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>gi|11995474|gb|NP_063937.1|GSK3A 483aa linear glycogen synthase kinase 3 alpha [Homo sapiens].
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NSS
>gi|7019350|gb|NM_013246.1|CLC 1689bp mRNA Homo sapiens cardiotrophin-like cytokine (CLC), mRNA.
GCCTCCGGGAGAGGAGCCGCACCCGGCCGGCCCGGCCCCAGCCCCATGGACCTCCGAGCA
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CTCACCCGCTACCTGGAGCACCAACTCCGCAGCTTGGCTGGGACCTATCTGAACTACCTG
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AGGGCCACTGTTGACTTGGAGGTGTGGCGAAGCCTCAATGACAAACTGCGGCTGACCCAG
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AACATGGTGGCAATTCTACACAAAAAGAGATGAGATTAACAGTGCAGGGTTGGGGTCTGC
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TTGCCCCAG
>gi|7019351|gb|NP_037378.1|CLC 225aa linear cardiotrophin-like cytokine; neurotrophin-1/B-cell stimulating factor-3 [Homo sapiens].
MDLRAGDSWGMLACLCTVLWHLPAVPALNRTGDPGPGPSIQKTYDLTRYLEHQLRSLAGT
YLNYLGPPFNEPDFNPPRLGAETLPRATVDLEVWRSLNDKLRLTQNYEAYSHLLCYLRGL

NRQAATAELRRSLAHFCTSLQGLLGSIAGVMAALGYPLPQPLPGTEPTWTPGPAHSDFLQ
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>gi|22068574|gb|XM_036493.3|ZNF213 3073bp mRNA Homo sapiens
zinc finger protein 213 (ZNF213), mRNA.
GGCCTCTGGCCGCCTGGCTCCAACATCAAGCACCGGGCTCCGAGTGGCCGGGATCAGCGC
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GCCCCGGCACCCTGCTCCATCGGCACTGGCGCCCTGCTCCATCGGCACTAATGCTCCACT
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TCCAACGGCACTGGTGCCCCATTCCATCGGCACTAACGCCCCGCTCCACCGGCACCAGTG
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CTAACGCCCTGCT

>gi|14777854|gb|XP_036493.1|ZNF213 459aa linear similar to Zinc finger protein 213 (Putative transcription factor CR53) [Homo sapiens].

MAAPLEAQDQAPGEGEGLLIVKVEDSSWEQESAQHEDGRDSEACRQRFRQFCYGDVHGPH
EAFSQLWELCCRWLRPELRTKEQILELLVLEQFLTVLPGEIQGWVREQHPGSGEEAVALV
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>gi|21536281|gb|NM_003656.3|CAMK1 1501bp mRNA Homo sapiens calcium/calmodulin-dependent protein kinase I (CAMK1), mRNA.

GGAGAGAGCCGCCGAGCCGAGCCGAGCCCCAGCTCCAGCAAGAGCGCGGGCGGGTGGCCC
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A
>gi|4502553|gb|NP_003647.1|CAMK1 370aa linear calcium/calmodulin-dependent protein kinase I [Homo sapiens].
MLGAVEGPRWKQAEDIRDIYDFRDVLGTGAFSEVILAEDKRTQKLVAIKCIAKEALEGKE
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TPGYVAPEVLAQKPYSKAVDCWSIGVIAYILLCGYPPFYDENDAKLFEQILKAEYEFDSP
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ELSPTLPHQL
>gi|13186237|gb|NM_023107.1|FGFR1 2590bp mRNA Homo sapiens fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome) (FGFR1), transcript variant 5, mRNA.
CCTCTTGCGGCCACAGGCGCGGCGTCCTCGGCGGCGGGCGGCAGCTAGCGGGAGCCGGGA
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GCCACTGCACTCCAGCCTGGGTGACAGAGTGAGTGAGACTCTGCGTTAAAAAAAAAAAAA
AAAAAAAAAA

>gi|13186238|gb|NP_075595.1|FGFR1 302aa linear fibroblast growth factor receptor 1 isoform 5 precursor; fms-related tyrosine kinase-2; heparin-binding growth factor receptor; FMS-like tyrosine kinase 2; basic fibroblast growth factor receptor 1; N-sam tyrosine kinase; FLG protein; protein-tyrosine kinase; tyrosylprotein kinase; hydroxyaryl-protein kinase [Homo sapiens].

MWSWKCLLFWAVLVTATLCTARPSPTLPEQDALPSSEDDDDDDSSSEEKETDNTKPNRM
PVAPYWTSPEKMEKKLHAVPAAKTVKFKCPSSGTPNPTLRWLKNGKEFKPDHRIGGYKVR
YATWSIIMDSVVPSDKGNYTCIVENEYGSINHTYQLDVVERSPHRPILQAGLPANKTVAL
GSNVEFMCKVYSDPQPHIQWLKHIEVNGSKIGPDNLPYVQILKVIMAPVFVGQSTGKETT
VSGAQVPVGRLSCPRMGSFLTLQAHTLHLSRDLATSPRTSNRGHKVEVSWEQRAAGMGGA
GL

>gi|4758007|gb|NM_004071.1|CLK1 1834bp mRNA Homo sapiens CDC-like kinase 1 (CLK1), mRNA.

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>gi|4758008|gb|NP_004062.1|CLK1 484aa linear CDC-like kinase 1; protein tyrosine kinase STY [Homo sapiens].

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KKSI

>gi|20127640|gb|NM_025128.2|MUS81 2352bp mRNA Homo sapiens MUS81 endonuclease (MUS81), mRNA.

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AAATAATAAAAATAAATAAAACTTCCTAAAAGAAAAGATTGAAACCCAAAAAAAAAAAAA
AAAAAAAAAAA

>gi|13376707|gb|NP_079404.1|MUS81 476aa linear MUS81 endonuclease [Homo sapiens].

MLDERLQRHRTSGGDHAPDSPSGENSPAPQGRLAEVQDSSMPVPAQPKAGGSGSYWPARH
SGARVILLVLYREHLNPNGHHFLTKEELLQRCAQKSPRVAPGSAPPWPALRSLLHRNLVL
RTHQPARYSLTPEGLELAQKLAESEGLSLLNVGIGPKEPPGEETAVPGAASAELASEAGV
QQQPLELRPGEYRVLLCVDIGETRGGGHRPELLRELQRLHVTHTVRKLHVGDFVWVAQET
NPRDPANPGELVLDHIVERKRLDDLCSSIIDGRFREQKFRLKRCGLERRVYLVEEHGSVH
NLSLPESTLLQAVTNTQVIDGFFVKRTADIKESAAYLALLTRGLQRLYQGHTLRSRPWGT
PGNPESGAMTSPNPLCSLLTFSDFNAGAIKNKAQSVREVFARQLMQVRGVSGEKAAALVD
RYSTPASLLAAYDACATPKEQETLLSTIKCGRLQRNLGPALSRTLSQLYCSYGPLT

>gi|19923239|gb|NM_003376.2|VEGF 3166bp mRNA Homo sapiens vascular endothelial growth factor (VEGF), mRNA.

AAGAGCTCCAGAGAGAAGTCGAGGAAGAGAGAGACGGGGTCAGAGAGAGCGCGCGGGCGT
GCGAGCAGCGAAAGCGACAGGGGCAAAGTGAGTGACCTGCTTTTGGGGGTGACCGCCGGA
GCGCGGCGTGAGCCCTCCCCCTTGGGATCCCGCAGCTGACCAGTCGCGCTGACGGACAGA
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GACGCGGCGGCGAGCCGCGGGCAGGGGCCGGAGCCCGCCCCCGGAGGCGGGGTGGAGGGG
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>gi|19923240|gb|NP_003367.2|VEGF 191aa linear vascular endothelial growth factor [Homo sapiens].

MNFLLSWVHWSLALLLYLHHAKWSQAAPMAEGGGQNHHEVVKFMDVYQRSYCHPIETLVD
IFQEYPDEIEYIFKPSCVPLMRCGGCSNDEGLECVPTEESNITMQIMRIKPHQGQHIGEM
SFLQHNKCECRPKKDRARQENPCGPCSERRKHLFVQDPQTCKCSCKNTHSRCKARQLELN
ERTCRCDKPRR

>gi|16306545|gb|NM_033649.1|FGF18 1466bp mRNA Homo sapiens fibroblast growth factor 18 (FGF18), transcript variant 2, mRNA.

CACGGCCGGAGAGACGCGGAGGAGGAGACATGAGCCGGCGGGCGCCCAGACGGAGCGGCC
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AAAAAAAAACAAAAAAAAAAAAAAAA
>gi|16306546|gb|NP_387498.1|FGF18 207aa linear fibroblast growth factor 18 precursor [Homo sapiens].
MYSAPSACTCLCLHFLLLCFQVQVLVAEENVDFRIHVENQTRARDDVSRKQLRLYQLYSR
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>gi|24496766|gb|NM_004712.3|HGS 2926bp mRNA Homo sapiens hepatocyte growth factor-regulated tyrosine kinase substrate (HGS), mRNA.
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GGTACAGCGATAATAAAATGTATTTCAGAAAAAAAAAAAAAAAAAA
>gi|4758528|gb|NP_004703.1|HGS 777aa linear hepatocyte growth factor-regulated tyrosine kinase substrate; human growth factor-regulated tyrosine kinase substrate [Homo sapiens].
MGRGSGTFERLLDKATSQLLLETDWESILQICDLIRQGDTQAKYAVNSIKKKVNDKNPHV
ALYALEVMESVVKNCGQTVHDEVANKQTMEELKDLLKRQVEVNVRNKILYLIQAWAHAFR
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>gi|20127435|gb|NM_003821.2|RIPK2 1898bp mRNA Homo sapiens receptor-interacting serine-threonine kinase 2 (RIPK2), mRNA.
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>gi|4506537|gb|NP_003812.1|RIPK2 540aa linear receptor-interacting serine-threonine kinase 2; receptor interacting protein 2 [Homo sapiens].
MNGEAICSALPTIPYHKLADLRYLSRGASGTVSSARHADWRVQVAVKHLHIHTPLLDSER
KDVLREAEILHKARFSYILPILGICNEPEFLGIVTEYMPNGSLNELLHRKTEYPDVAWPL
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SKSAPEGGTIIYMPPENYEPGQKSRASIKHDIYSYAVITWEVLSRKQPFEDVTNPLQIMY
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SRSLPAPQDNDFLSRKAQDCYFMKLHHCPGNHSWDSTISGSQRAAFCDHKTTPCSSAIIN
PLSTAGNSERLQPGIAQQWIQSKREDIVNQMTEACLNQSLDALLSRDLIMKEDYELVSTK
PTRTSKVRQLLDTTDIQGEEFAKVIVQKLKDNKQMGLQPYPEILVVSRSPSLNLLQNKSM

>gi|26051238|gb|NM_021137.3|TNFAIP1 3571bp mRNA Homo sapiens tumor necrosis factor, alpha-induced protein 1 (endothelial) (TNFAIP1), mRNA.

CACAGCTTGGGACTGCTGAGGGGCAGGCGGCTGCAGGCTAGGGGCGGCTCGGAGTCCGCT
GGCCACCCAGCTGAGAGGAGAGGCGCCCCCGGGGACGCACTGAGATTATGAGGCTCTGGC
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>gi|10863937|gb|NP_066960.1|TNFAIP1 316aa linear tumor necrosis factor, alpha-induced protein 1 [Homo sapiens].

MSGDTCLCPASGAKPKLSGFKGGGLGNKYVQLNVGGSLYYTTVRALTRHDTMLKAMFSGR
MEVLTDKEGWILIDRCGKHFGTILNYLRDDTITLPQNRQEIKELMAEAKYYLIQGLVNMC
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FPEARIYEETLNVLLYETPRVPDNSLLEATSRSRSQASPSEDEETFELRDRVRRIHVKRY
STYDDRQLGHQSTHRD

>gi|27597077|gb|NM_006293.2|TYRO3 3949bp mRNA Homo sapiens TYRO3 protein tyrosine kinase (TYRO3), mRNA.

GCGGTGGCGCGGGAGCGGCCCCGGGGACCCCGCGCTGCTGACGGCGGCGACCGCGGCCGG
AGGCGGGCGCGGGTCTCGGAGGCGGTCGCCTCAGCACCGCCCCACGGGCGGCCCCAGCCC
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CCTCGCTCGCGGGCCGGGCCCGGCATGGTGCGGCGTCGCCGCCGATGGCGCTGAGGCGGA
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>gi|27597078|gb|NP_006284.2|TYRO3 890aa linear TYRO3 protein tyrosine kinase; Brt; Dtk; Sky; Tif; Tyro3 protein tyrosine

kinase (sea-related receptor tyrosine kinase); tyrosine-protein kinase receptor TYRO3 precursor [Homo sapiens].
MALRRSMGRPGLPPLPLPPPPRLGLLLAALASLLLPESAAAGLKLMGAPVKLTVSQGQPV
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ASVAWMPGADGRALLQSCTVQVTQAPGGWEVLAVVVPVPPFTCLLRDLVPATNYSLRVRC
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DGSFVKVAVKMLKADIIASSDIEEFLREAACMKEFDHPHVAKLVGVSLRSRAKGRLPIPM
VILPFMKHGDLHAFLLASRIGENPFNLPLQTLIRFMVDIACGMEYLSSRNFIHRDLAARN
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CLRMELENILGQLSVLSASQDPLYINIERAEEPTAGGSLELPGRDQPYSGAGDGSGMGAV
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>gi|4502884|gb|NM_003992.1|CLK3 1762bp mRNA Homo sapiens CDC-like kinase 3 (CLK3), transcript variant phclk3, mRNA.
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AAAGTGTTTCTTACTGTTTGTA
>gi|4502885|gb|NP_003983.1|CLK3 490aa linear CDC-like kinase 3
isoform hclk3 [Homo sapiens].
MHHCKRYRSPEPDPYLSYRWKRRRSYSREHEGRLRYPSRREPPPRRSRSRSHDRLPYQRR
YRERRDSDTYRCEERSPSFGEDYYGPSRSRHRRRSRERGPYRTRKHAHHCHKRRTRSCSS
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SFHTSRNPSR
>gi|9910121|gb|NM_020249.1|ADAMTS9 3674bp mRNA Homo sapiens a
disintegrin-like and metalloprotease (reprolysin type) with
thrombospondin type 1 motif, 9 (ADAMTS9), mRNA.
GCGGGAAGCACCATGCAGTTTGTATCCTGGGCCACACTGCTAACGCTCCTGGTGCGGGAC
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>gi|9910122|gb|NP_064634.1|ADAMTS9 1072aa linear a disintegrin and metalloproteinase with thrombospondin motifs-9 preproprotein [Homo sapiens].

MQFVSWATLLTLLVRDLAEMGSPDAAAAVRKDRLHPRQVKLLETLGEYEIVSPIRVNALG
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>gi|17981697|gb|NM_001262.2|CDKN2C 2104bp mRNA Homo sapiens cyclin-dependent kinase inhibitor 2C (p18, inhibits CDK4) (CDKN2C), transcript variant 1, mRNA.
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AAAA
>gi|4502751|gb|NP_001253.1|CDKN2C 168aa linear cyclin-dependent kinase inhibitor 2C; cyclin-dependent kinase 6

inhibitor p18; cyclin-dependent kinase 4 inhibitor C; cyclin-dependent inhibitor; CDK6 inhibitor p18 [Homo sapiens].
MAEPWGNELASAAARGDLEQLTSLLQNNVNVNAQNGFGRTALQVMKLGNPEIARRLLLRG
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>gi|23510344|gb|NM_002037.3|FYN 2650bp mRNA Homo sapiens FYN oncogene related to SRC, FGR, YES (FYN), transcript variant 1, mRNA.
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CAAAAACCAA
>gi|4503823|gb|NP_002028.1|FYN 537aa linear protein-tyrosine kinase fyn isoform a; proto-oncogene tyrosine-protein kinase fyn; src/yes-related novel gene; src-like kinase; c-syn protooncogene; tyrosine kinase p59fyn(T); OKT3-induced calcium influx regulator [Homo sapiens].
MGCVQCKDKEATKLTEERDGSLNQSSGYRYGTDPTPQHYPSFGVTSIPNYNNFHAAGGQG
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>gi|15055546|gb|NM_000800.2|FGF1 2357bp mRNA Homo sapiens fibroblast growth factor 1 (acidic) (FGF1), transcript variant 1, mRNA.
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AAAAAAAAAAAAAAAAA

>gi|4503697|gb|NP_000791.1|FGF1 155aa linear fibroblast growth factor 1 (acidic) isoform 1 precursor; heparin-binding growth factor 1 precursor; endothelial cell growth factor, alpha; endothelial cell growth factor, beta [Homo sapiens].

MAEGEITTFTALTEKFNLPPGNYKKPKLLYCSNGGHFLRILPDGTVDGTRDRSDQHIQLQ
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NWFVGLKKNGSCKRGPRTHYGQKAILFLPLPVSSD

>gi|27552761|gb|NM_002825.3|PTN 1029bp mRNA Homo sapiens pleiotrophin (heparin binding growth factor 8, neurite growth-promoting factor 1) (PTN), mRNA.

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>gi|4506281|gb|NP_002816.1|PTN 168aa linear pleiotrophin (heparin binding growth factor 8, neurite growth-promoting

factor 1); heparin affin regulatory protein; heparin-binding growth-associated molecule [Homo sapiens].
MQAQQYQQQRRKFAAAFLAFIFILAAVDTAEAGKKEKPEKKVKKSDCGEWQWSVCVPTSG
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>gi|4504008|gb|NM_000169.1|GLA 1350bp mRNA Homo sapiens galactosidase, alpha (GLA), mRNA.
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>gi|4504009|gb|NP_000160.1|GLA 429aa linear galactosidase, alpha [Homo sapiens].
MQLRNPELHLGCALALRFLALVSWDIPGARALDNGLARTPTMGWLHWERFMCNLDCQEEP
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ANYVHSKGLKLGIYADVGNKTCAGFPGSFGYYDIDAQTFADWGVDLLKFDGCYCDSLENL
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RHISPQAKALLQDKDVIAINQDPLGKQGYQLRQGDNFEVWERPLSGLAWAVAMINRQEIG
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MQMSLKDLL
>gi|18587778|gb|XM_091624.1|LOC162542 287bp mRNA Homo sapiens similar to ADP-ribosylation factor 1 (LOC162542), mRNA.
GTCTGATTTTTATGGTTGACAGTAATGACAGAGAGCAGATTGATGAGGCCTGGGAAGTGC
TAACTTACTTGTTAGAGGACGATGAGCTCAGAAATGCAGTTTTATTGGTATTTGCCAATA
AACAAGATCTCCCTAATACTATGAACGCGGCAGAGATAACGGACAAGCTCGGCCTCCATT
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ACGAAGGCCTGAACTGGCTCGCCAACCAGTTCCAGAACCAGAACTGA

>gi|18587779|gb|XP_091624.1|LOC162542 91aa linear similar to ADP-ribosylation factor 1 [Homo sapiens].
MVDSNDREQIDEAWEVLTYLLEDDELRNAVLLVFANKQDLPNTMNAAEITDKLGLHSLRY
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>gi|4557572|gb|NM_000401.1|EXT2 3781bp mRNA Homo sapiens exostoses (multiple) 2 (EXT2), mRNA.
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T

>gi|4557573|gb|NP_000392.1|EXT2 718aa linear exostoses (multiple) 2 [Homo sapiens].

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>gi|27597083|gb|NM_006838.2|METAP2 1908bp mRNA Homo sapiens methionyl aminopeptidase 2 (METAP2), mRNA.

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>gi|5803092|gb|NP_006829.1|METAP2 478aa linear methionyl aminopeptidase 2; methionine aminopeptidase; eIF-2-associated p67 [Homo sapiens].

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>gi|10864040|gb|NM_021230.1|MLL3 12689bp mRNA Homo sapiens myeloid/lymphoid or mixed-lineage leukemia3 (MLL3), mRNA.

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>gi|10864041|gb|NP_067053.1|MLL3 4025aa linear myeloid/lymphoid or mixed-lineage leukemia 3; ALR-like protein [Homo sapiens].

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>gi|21359851|gb|NM_000966.2|RARG 2663bp mRNA Homo sapiens retinoic acid receptor, gamma (RARG), mRNA.

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>gi|4506423|gb|NP_000957.1|RARG 454aa linear retinoic acid receptor, gamma; Retinoic acid receptor, gamma polypeptide [Homo sapiens].

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>gi|6978649|gb|NP_005189.2|CHKL 395aa linear choline/ethanolamine kinase isoform a [Homo sapiens].

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MSTVHEILCKLSLEGDHSTPPSAYGSVKAYTNFDAERDALNIETAIKTKGVDEVTIVNIL
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GT
>gi|18578340|gb|XP_084635.1|LOC143785 211aa linear similar to hypothetical protein XP_084635 [Homo sapiens].
MVFLKFFCMSFFCHLCQGYFDGPLYPEMSNGTLHHYFVPDGDYEENDDPEKCQLLFRVSD
HRRCSQGEGSQVGSLLSLTLREEFTVLGRQVEDAGRVLEGISKSISYDLDGEESYGKYLR
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>gi|4507464|gb|NM_003239.1|TGFB3 2574bp mRNA Homo sapiens transforming growth factor, beta 3 (TGFB3), mRNA.
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>gi|4507465|gb|NP_003230.1|TGFB3 412aa linear transforming growth factor, beta 3 [Homo sapiens].
MKMHLQRALVVLALLNFATVSLSLSTCTTLDFGHIKKKRVEAIRGQILSKLRLTSPPEPT
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>gi|21735553|gb|NM_002419.2|MAP3K11 3603bp mRNA Homo sapiens mitogen-activated protein kinase kinase kinase 11 (MAP3K11), mRNA.
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AAA

>gi|4505195|gb|NP_002410.1|MAP3K11 847aa linear mitogen-activated protein kinase kinase kinase 11; mixed lineage kinase 3; SH3 domain-containing proline-rich kinase; protein-tyrosine kinase PTK1 [Homo sapiens].

MEPLKSLFLKSPLGSWNGSGSGGGGGGGGRPEGSPKAAGYANPVWTALFDYEPSGQDEL
ALRKGDRVEVLSRDAAISGDEGWWAGQVGGQVGIFPSNYVSRGGGPPPCEVASFQELRLE
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WVPEAGP

>gi|4505784|gb|NM_000294.1|PHKG2 1571bp mRNA Homo sapiens phosphorylase kinase, gamma 2 (testis) (PHKG2), mRNA.

AAGGTGAGCGACTGCAGGCAAACCCGGCGACAGCGCAGCTCGCGTCGACCCTGGCTCCTC
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ACACGCCAGGG

>gi|4505785|gb|NP_000285.1|PHKG2 406aa linear phosphorylase kinase, gamma 2 (testis); Phosphorylase kinase, gamma 2 (testis/liver) [Homo sapiens].

MTLDVGPEDELPDWAAAKEFYQKYDPKDVIGRGVSSVVRRCVHRATGHEFAVKIMEVTAE
RLSPEQLEEVREATRRETHILRQVAGHPHIITLIDSYESSSFMFLVFDLMRKGELFDYLT
EKVALSEKETRSIMRSLLEAVSFLHANNIVHRDLKPENILLDDNMQIRLSDFGFSCHLEP
GEKLRELCGTPGYLAPEILKCSMDETHPGYGKEVDLWACGVILFTLLAGSPPFWHRRQIL
MLRMIMEGQYQFSSPEWDDRSSTVKDLISRLLQVDPEARLTAEQALQHPFFERCEGSQPW
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>gi|5453790|gb|NP_006160.1|NNMT 264aa linear nicotinamide N-methyltransferase [Homo sapiens].
MESGFTSKDTYLSHFNPRDYLEKYYKFGSRHSAESQILKHLLKNLFKIFCLDGVKGDLLI
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RVKGPEKEEKLRQAVKQVLKCDVTQSQPLGAVPLPPADCVLSTLCLDAACPDLPTYCRAL
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SYSSTMANNEGLFSLVARKLSRPL
>gi|4507668|gb|NM_003295.1|TPT1 830bp mRNA Homo sapiens tumor protein, translationally-controlled 1 (TPT1), mRNA.
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>gi|4507669|gb|NP_003286.1|TPT1 172aa linear tumor protein, translationally-controlled 1; fortilin; histamine-releasing factor [Homo sapiens].
MIIYRDLISHDEMFSDIYKIREIADGLCLEVEGKMVSRTEGNIDDSLIGGNASAEGPEGE
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>gi|27477074|gb|NP_061195.2|IL17BR 502aa linear IL-17B receptor isoform 1 precursor; IL-17B receptor; interleukin 17 receptor homolog 1; interleukin 17 receptor homolog; cytokine receptor CRL4 [Homo sapiens].

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>gi|14165275|gb|NM_032411.1|ECRG4 772bp mRNA Homo sapiens esophageal cancer related gene 4 protein (ECRG4), mRNA.

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cancer related gene 4 protein [Homo sapiens].
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